T H E S I S

presented for

the Degree of Doctor of Medicine

in the University of Edinburgh

entitled

PEPTIC ULCERATION OF THE OESOPHAGUS

IN COMBINATION WITH

PARTIAL THORACIC STOMACH

by

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September, 1944.

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I N D E X

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PREFACE

I have the honour to present this thesis for the Doctorate of Medicine of the University of Edinburgh.

The subject - "Peptic Ulceration of the Oesophagus in combination with Partial Thoracic Stomach" - is one which has been recognised only within the past few years. It has been my fortune, as radiologist to a large teaching hospital, to find over twenty cases during the past five years. The thesis reviews the pathogenesis and clinical features together with a description of diagnostic methods and treatment. The appendix contains full clinical notes of eighteen cases which have been under treatment and observation for more than a year.

The clinical importance in recognising the condition lies in the fact that the alternative diagnosis is cancer of the lower end of the oesophagus. Peptic ulceration is not uncommon and if erroneously treated by radiotherapy the results might prove disastrous.

The academic interest of the subject centres round the lower end of the oesophagus and the partial thoracic stomach. The congenital and acquired types of gastric herniation through the oesophageal hiatus are fully discussed and the accepted anatomical site of the cardia is called

into question. As regurgitation into the oesophagus plays a large part in the production of peptic ulceration this point assumes considerable importance. The recognition of the position of the cardia in relation to neighbouring structures is most readily determined by radiology and stress is laid on the distinguishing features.

I should like to acknowledge my great indebtedness to my colleague, Mr. P.R. Allison, F.R.C.S., for placing at my disposal his clinical notes and for the stimulating interest he has shown in the production of this thesis. Free use has been made of earlier publications by myself in association with Mr. Allison.

To my colleagues in the Radiology Department, to Professor Stewart, Dr. Horne and Dr. Stanbury, to Miss Brown and Miss Milne for their excellent drawings and to Dr. J.W. Orr for his most helpful criticism, my grateful thanks are due.

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September 1944. The General Infirmary at Leeds.

HISTORICAL

Peptic ulceration of the oesophagus has long been recognised by pathologists but it is only in recent years that clinicians have identified it as an entity with a characteristic syndrome.

Quincke (1879) was the first to publish a case histologically proved. In 1906 Tileston recorded forty-four cases of peptic ulceration of the oesophagus. Stewart and Hartfall (1929) considered only nine of these to be true chronic ulcers and they collected ten others after an extensive review of the literature. They added one case of their own.

In the same year Friedenwald, Feldman and Zinn reported thirteen cases and Chevalier Jackson twenty-one cases which he had found in the course of 4,000 oesophagoscopies. He also described sixty-seven cases in which scars showed evidence of previous ulceration.

In 1934 Haroen and Gerlings published a case of a child of three years who was found to have an ulcer of the lower end of the oesophagus with a diaphragmatic hernia of the stomach and a congenital short oesophagus.

In his Arris and Gale Lecture, Dunhill (1934) mentions two cases of diaphragmatic hernia of the stomach with stricture at the lower end of the oesophagus. Endoscopy showed some ulceration to be present, just above the narrowing.

It was not until 1939, however, that the combination of peptic ulceration of the oesophagus with a congenitally short oesophagus and diaphragmatic hernia was described as a clinical entity. In this country Dick and Hurst (1939) described eight cases at the Royal Society of Medicine and simultaneously in America Feldman published two cases. In 1943 Allison, Johnstone and Royce described ten cases together with a full review of the pathogenesis, clinical features and treatment. Dick and Hurst subsequently (1943) added eight cases to their original contribution describing in detail the clinical syndrome, pathogenesis and treatment.

AETIOLOGY

SEX - Below is a tabulated list illustrating the sex incidence given by various authors.

AUTHOR	MALE	FEMALE
STEWART AND HURST	15	4
LYALL	5	3
FRIEDENWALD, FELDMAN AND ZINN ..	6	7
DICK AND HURST	4	12
ALLISON, JOHNSTONE AND ROYCE ..	5	5
ALLISON AND JOHNSTONE	5	2
TOTAL	40	33

When these figures are combined there are forty men and thirty-three women. It is fair to assume that the sex

incidence is almost equal with a slight preponderance in favour of men.

AGE

The following table gives the ages of published cases.

	20-29	30-39	40-49	50-59	60-69	70-79	80-89
DICK AND HURST	-	1	1	5	6	3	-
FRIEDENWALD, FELDMAN AND ZINN	1	2	4	5	1	-	-
ALLISON ET AL.	-	-	-	5	10	1	1

It is evident from these records that thirty-two out of forty-six cases (seventy per cent) occur in the 50-59 and 60-69 age groups.

MORBID ANATOMY

According to Dick and Hurst (1943) the morbid anatomy of chronic peptic ulcer of the oesophagus differs in no way from that of chronic gastric or duodenal ulcer. The ulcer is generally single, situated in the lower third of the oesophagus, usually just above the 'sphincter.'

In size it may be from a few millimetres in diameter to a lesion completely encircling the lumen. Small ulcers are round or oval; larger ones tend to be irregular and annular.

The wall is penetrated to a variable depth, with exposure or perforation of the muscular coat. The margins are usually clear-cut. From the floor and margins fibrosis extends outwards to involve adjacent structures. The presence of scars which are found more frequently than active ulcers indicate that these ulcers can heal completely.

Lyall (1937), in a careful analysis of eight advanced cases, described two types of ulcer; a diffuse superficial inflammation with areas of healing, sometimes intermingled with more chronic, deeper areas and a circumscribed deep ulcer. One striking feature was that in all ulcers the lower margin was sharply defined and within 1 cm. of the cardiac orifice whereas the upper limit was irregular. In one case the ulcer extended upwards in the form of two processes with a tongue of oesophageal mucous membrane between them. He described areas of regenerating epithelium "like thin patches of white paint on a raw surface."

PATHOGENESIS

(1) SEPSIS

Jackson (1929) laid particular stress on the influence of oral sepsis in producing oesophageal ulceration as a focus of infection was found in the tonsil, nasal sinuses or teeth in ninety per cent of his series of eighty-eight cases,

active or healed. In a series of eighty-eight control patients over the age of eighteen, oesophagoscoped for the removal of foreign bodies and in whom there was no peptic ulceration, only twelve per cent had infective foci.

Dick and Hurst (1943) note that more or less dental sepsis was present in all cases, save one who was edentulous. They remark that the absence or insufficiency of teeth is likely to cause irritation of the oesophagus due to inadequate mastication and this, they feel, is more important than dental sepsis.

In this series dental sepsis was frequently observed but the records regarding the condition of the air sinuses and tonsils are incomplete.

(2) HETEROTOPIC GASTRIC MUCOSA

Taylor (1922) described a series of autopsies where epithelial heterotopia was found in various parts of the alimentary tract proximal to the ileo-caecal valve. Six examples of gastric mucosa in the oesophagus were found in a consecutive series of 900 autopsies. These areas occurred just below the level of the cricoid cartilage. They varied in size from $\frac{1}{8}$" to $\frac{3}{4}$" in diameter and were usually bilateral and unequal in size. Microscopically the structure of fundal mucosa was faithfully reproduced,

the glands possessing both chief and oxyntic cells. Their rapid post-mortem digestion indicated that they secreted active gastric juice (Nicholson 1922).

Taylor found neither ulceration nor inflammatory reaction in relation to them and he believed that the secretion would be rapidly neutralised by saliva and extremely unlikely to cause damage to adjacent epithelium. Dick and Hurst consider it possible for sufficient acid juice to collect at the lower end and bring about a chronic peptic ulcer just above the cardia. Stewart and Hartfall (1929) described a case of peptic ulceration of the oesophagus where perforation had occurred and death had followed infection of the pleural cavity. Autopsy showed a large perforated chronic ulcer on the posterior wall of the oesophagus just above the gastro-oesophageal junction and, exactly opposite, on the anterior wall there was a large well-healed oval scar. At the upper part of the oesophagus just below the level of the cricoid cartilage there were two symmetrically disposed areas of heterotopic gastric mucosa.

On the other hand, Jackson (1929) recorded the presence of islets of gastric mucosa at the lower end of the oesophagus in a third of twenty-two cases in which oesophagoscopy had shown ulceration.

Brown Kelly (1939) described a series of cases occurring in children where islets of gastric mucosa had ulcerated giving rise to an oesophagitis. Schridde (1904) found that in seventy per cent of a series of autopsies glands of gastric mucosal type were found in the lower end of the oesophagus, though they did not appear mature.

It is conceivable that acid gastric juice secreted by gastric mucosa situated in the oesophagus may accumulate at the lower end of the oesophagus. Small areas of gastric heterotopia in the postcricoid region would easily be missed at endoscopy so that clinical material is unlikely to throw any light on its association with peptic ulcer. Although gastric heterotopia has frequently been found endoscopically at the lower end of the oesophagus in close association with peptic ulcer, Allison (1943) stresses the need for some caution in drawing conclusions about its aetiological significance. If the areas were true congenital misplacements they might have a causal relationship to ulceration. On the other hand, where extensive ulceration and scarring occur, the gastric mucosa may be drawn up irregularly and areas might even become separated from the rest of the stomach and remain as islands in the oesophageal wall. Thus, heterotopia may be the result, rather than the cause, of ulceration.

(3) THE ACID FACTOR

Chronic ulcers of the type found in the stomach and duodenum occur in those parts of the alimentary tract which are exposed for long periods to the action of gastric juice. They are constantly found in the distal two-thirds of the stomach and in the duodenal bulb. After gastro-enterostomy or partial gastrectomy they develop at or adjacent to the anastomosis if free hydrochloric acid is present. It is then reasonable to suppose that chronic ulcers at the lower end of the oesophagus are of peptic origin and are due to the unaccustomed presence of gastric juice containing free hydrochloric acid. Here it is of significance to note that ulceration of this type never occurs in association with achalasia in spite of the presence of chronic oesophagitis (Dick and Hurst.) The mixture of food, saliva and mucus in the dilated oesophagus is always alkaline and acid regurgitation does not occur. The frequency with which duodenal or gastric ulceration accompanies or precedes these cases of ulceration, makes it apparent that there is a special liability of patients with constitutional hyperchlorhydria to suffer.

Dick and Hurst found that four out of five patients who were given a test meal had hyperchlorhydria. Also

out of twenty-four cases of oesophageal ulceration found at autopsy, ten had duodenal or gastric ulceration.

In Allison's cases - except in one man who had a high normal curve - no information about gastric secretion is available as the swallowing of a Ryle's tube was prevented by the stenosis.

(4) REGURGITATION OF GASTRIC JUICE.

The regurgitation of acid gastric juice is perhaps the most important factor in the production of chronic peptic ulcer of the oesophagus. Briggs, Dick and Hurst (1939) were the first to draw attention to the association of oesophageal ulceration and congenital short oesophagus. On the other hand, Jackson in his publication (1929) denied there was any coexisting oesophageal abnormality. He wrote: "None of our patients were found to have abnormal patulousness at either the diaphragmatic pinch-cock or the cardia." This difference of opinion may in some measure be due to the difficulty in the recognition of shortening of the oesophagus for it is primarily a radiological diagnosis.

Allison, Johnstone and Royce (1943) in a summary of their cases stressed the possibility of the oesophageal shortening being acquired, following cicatrisation from chronic ulceration. They considered that in the

majority of cases the hiatus hernia, giving rise to free regurgitation, was probably acquired and not congenital.

In a second publication Dick and Hurst (1943) vigorously attacked this view giving various reasons why they considered it unacceptable.

The argument has considerable academic interest. For clarity, it is proposed first to examine in detail the anatomy of the cardia and the types of hiatus herniae and then review the theories put forward on each side.

ANATOMY OF THE CARDIA

At the lower end a portion of the thoracic oesophagus, 1 - 1.5 cms. in length, passes through the oesophageal opening in the diaphragm and is connected by a considerable amount of strong fibrous tissue to the boundaries of the opening. Above and in front the hiatus is bounded by the central tendon and a few decussating fibres of the right crus.

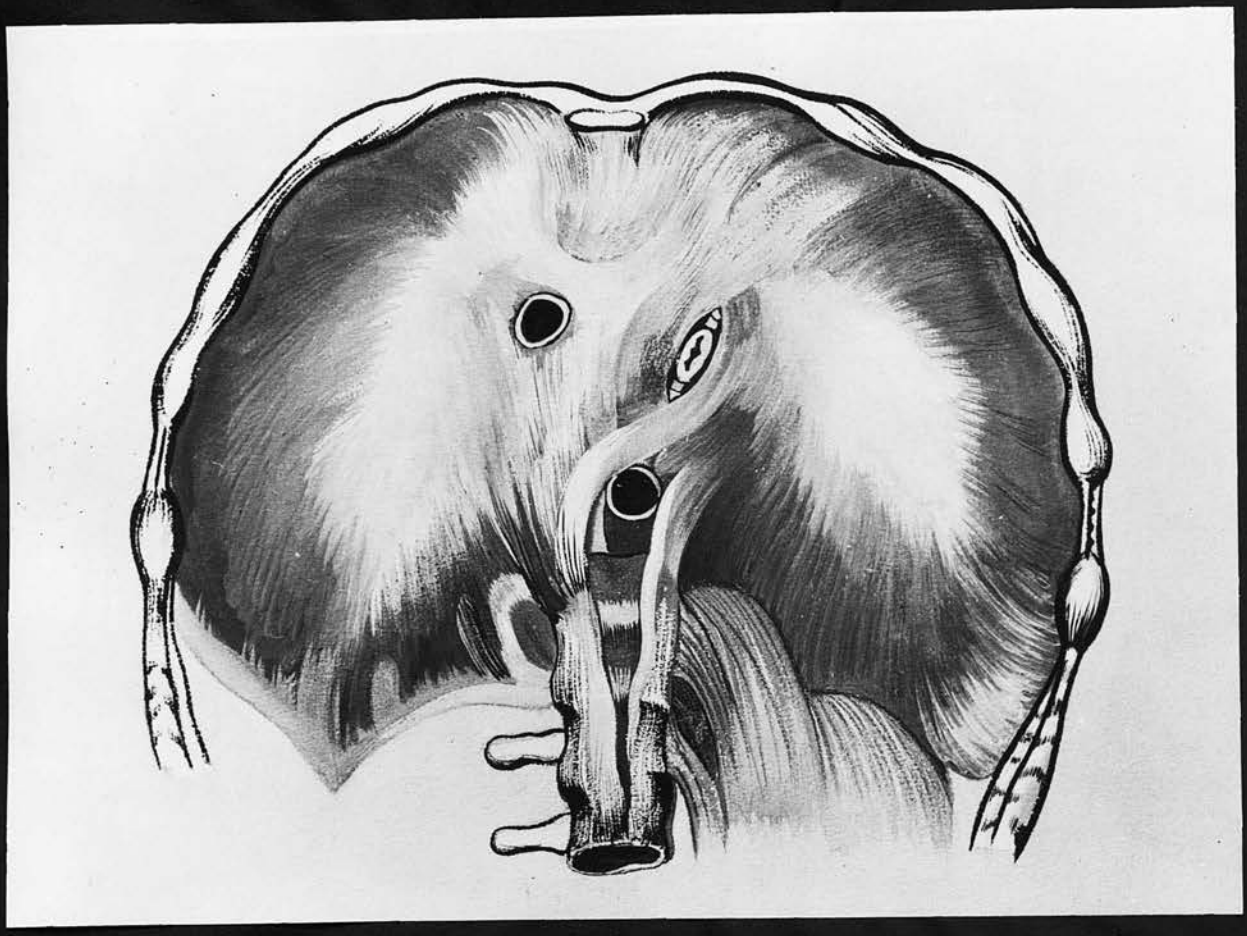
At the sides and behind, decussating bands from the right crus embrace the oesophagus and turn a flat surface - not an edge - towards the opening and thus behind and at the side the oesophagus is in contact with the diaphragm for a distance of 1 - 1.5 cms.

This segment is known as the ampulla phrenica. As the oesophagus enters the abdomen it inclines forwards and to the left for 1.5 - 2 cms. when it merges almost imperceptibly into the stomach. The intra-abdominal portion is known as the cardiac antrum or ampulla cardiaca and lies in the oesophageal groove on the posterior surface of the left lobe of the liver. Peritoneum covers it anteriorly and extends to its left side. (Fig. 1).

A more detailed description given by Lendrum (1937) is as follows:-

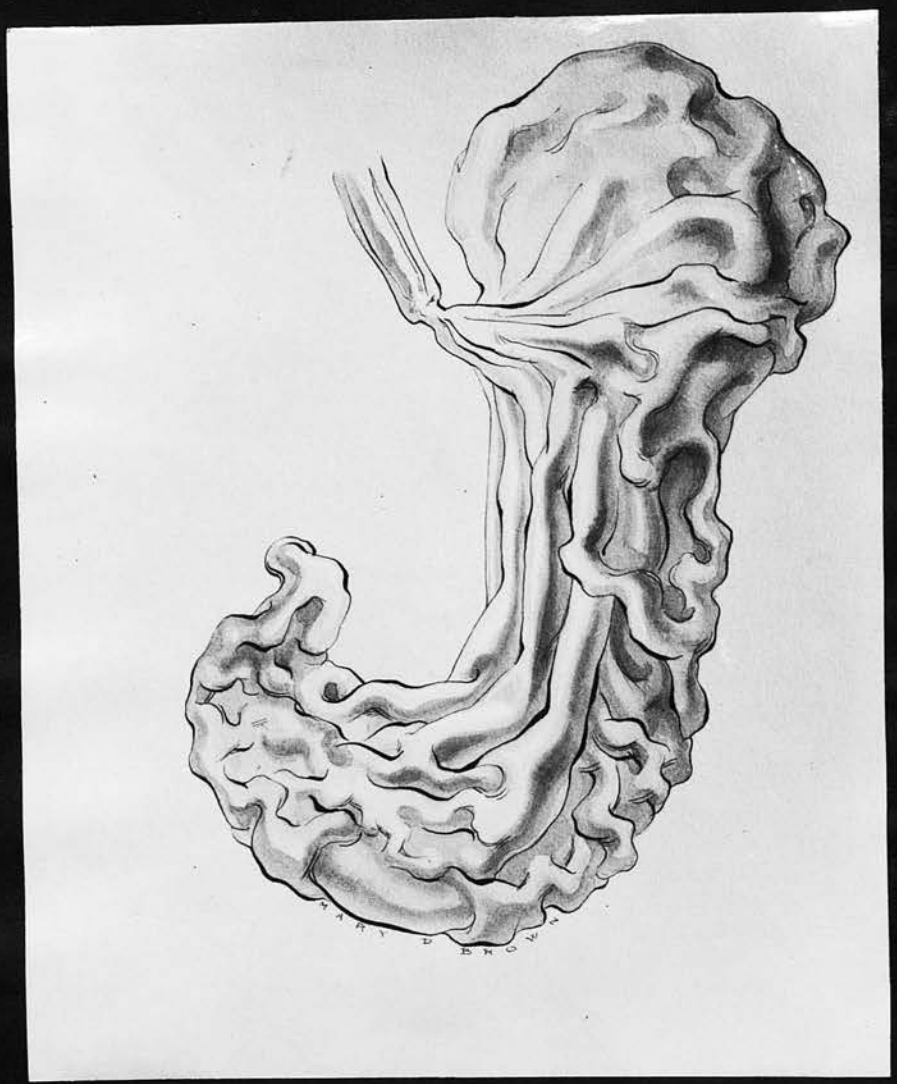
> "After passing through the diaphragm the lower end of the oesophagus curves to the left and enters the inner aspect of the upper end of the stomach at the cardiac orifice. In the human adult the abdominal portion of the oesophagus is about 2 cms. in length. At the cardiac orifice the right margin of the oesophagus is continuous with the lesser curvature of the stomach while the left margin joins the greater curvature at an acute angle which is termed the incisura cardiaca. This angulation depends on the form and position of the adjoining fundus.
>
> "The circular muscle fibres of the cardia differ from those in the remainder of the oesophagus in their relationship to the oblique muscle of the stomach. The oblique muscle is regarded as a special group of inner fibres of the circular muscle of the stomach. It does not form a complete layer but, on the contrary, is a ∩-shaped band with the curve passing over the incisura cardiaca and the limbs extending down the lesser curvature on either side

Fig. 1



Drawing of the inferior surface of the diaphragm, showing fibres of the right crus decussating round the oesophageal hiatus.
(From Gray's Anatomy.)

Fig. 2

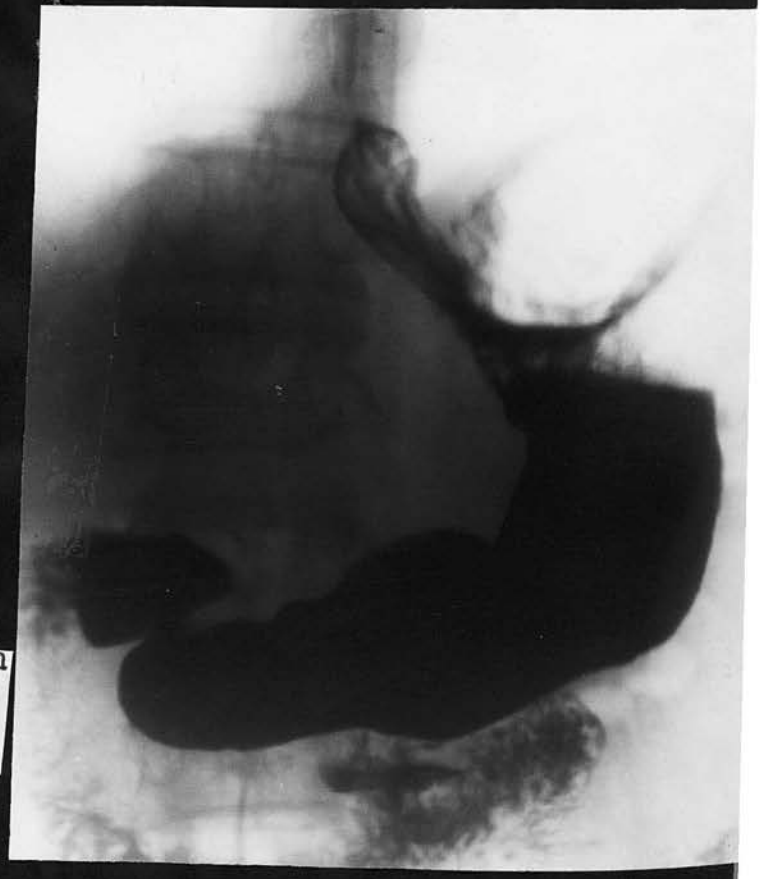


Artist's drawing of the stomach as described by the author. The nipple-like projection of gastric mucosa into the ampulla cardiaca is well illustrated.

Fig. 3

Erect. Normal stomach in a woman of 35. Coarse mucosal folds are seen running from the stomach into the lower oesophagus.

Fig. 4



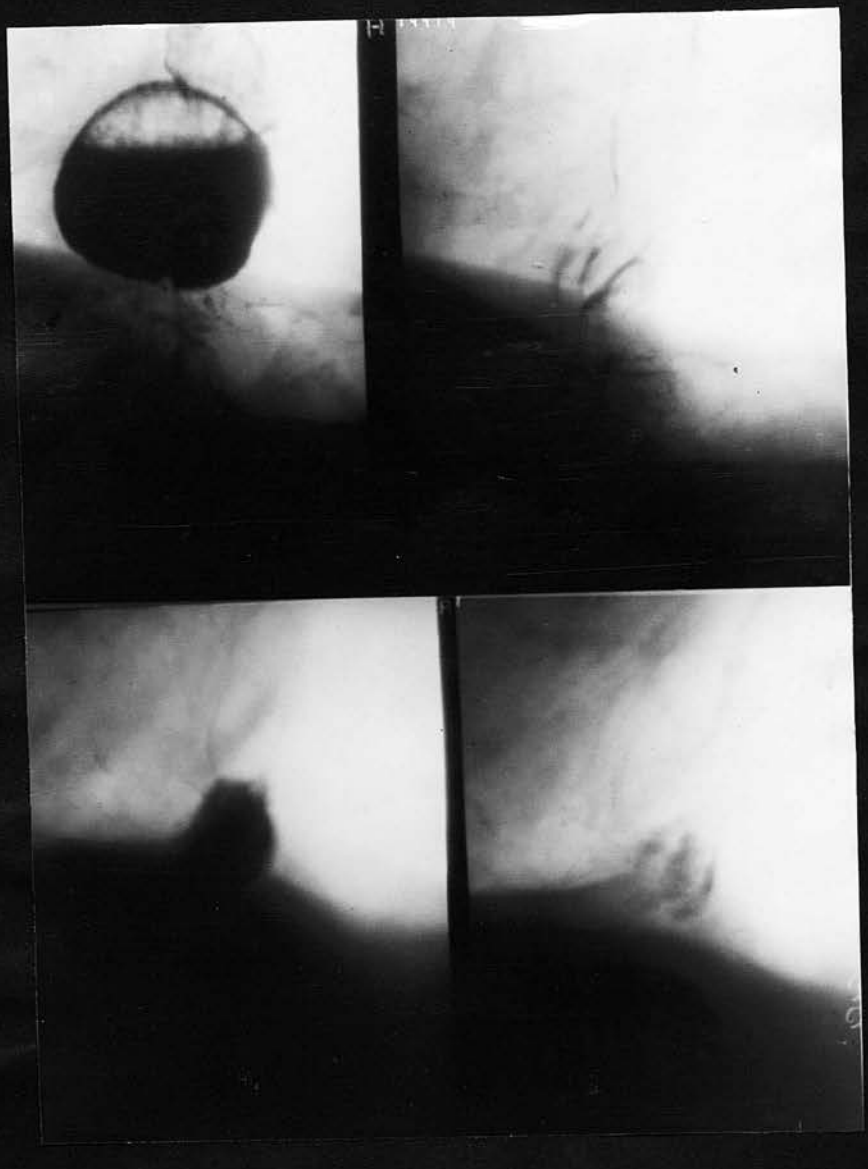
Erect. Shows stomach in man of 40. Larger nipple-like gastric pouch, amounting to pulsion hiatus hernia.

> "of the magenstrasse. These fibres loop up into the oesophagus for 1 cm. and blend with the circular fibres on the left aspect of the cardia."

Lendrum further suggests that overaction of the oblique muscle may exert a pinchcock action on the cardiac orifice.

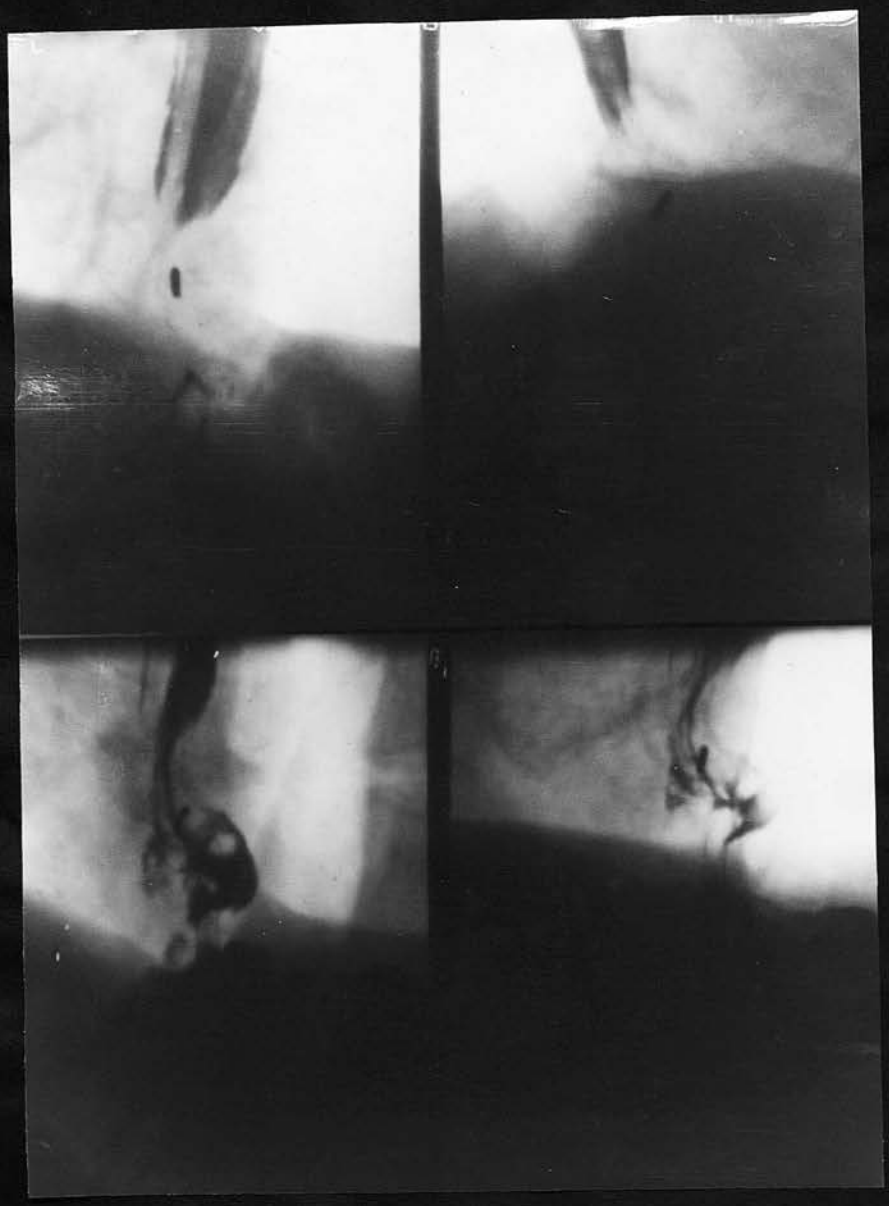
The junction of the squamous-and columnar-celled epithelium is usually a little irregular and its exact site in relation to the gastric wall is difficult to determine. The impression is gained that it extends further up the oesophagus than is realised or, in other words, at the cardia the stomach has a nipple-like projection of gastric mucosa (Figs. 2-4). It has probably a moderate range of movement which is only to be expected when one considers the stresses imposed on this junction by respiration and alterations in the position of the stomach. Radiologically the change from oesophageal to gastric mucosa is often clearly recognised by the alteration in mucosal pattern and the accuracy of this observation has been tested and proved on a number of occasions by a method devised by Allison. He designed a special pair of forceps with which a silver Cushing brain clip can be attached to the cardio-oesophageal junction. Oesophagoscopy is performed, the junction of oesophageal and gastric mucous membrane identified and grasped with biopsy forceps. A small fold of mucosa is made, and a clip attached to it, the piece held

Fig. 5



Four radiographs showed mucosal changes at the lower end of the oesophagus which suggested that a small hiatus hernia of the stomach was present.

Fig. 6



TOP. Left and Right. Clip applied by Allison at site of biopsy. Section showed junctional mucosa.

LOWER. Left and Right. Supine position. Small gastric pouch outlined with barium. Varicose mucosal folds clearly demonstrated.

by the biopsy forceps is then removed for histological examination. Figs.5 and 6 show the radiological appearance of such a case after application of clips. The biopsy contained both oesophageal and gastric mucous membrane.

To substantiate this hypothesis of the mobility of the cardia some interesting observations were made in 1931 by Findlay and Brown Kelly. They suggested that the movement of the cardia through the hiatus was a physiological phenomenon. They had previously reported nine cases of dysphagia in children between the ages of one and ten, in whom they found an oesophageal stenosis and were struck by the wide shadow below the stenosis seen radiologically. Before realising, as a result of histology, that this was due to the filling of a gastric pouch, they naturally thought they were dealing with a dilated oesophagus and tried, by a similar technique, to produce the same shadow in normal children. Their method was to fill the stomach with fluid barium and then screen or take serial films while the child swallowed thick paste.

They were successful in their experiment but were astonished when they found that after taking sections from the centre of the dilatation, which by measurements was considered to be certainly supradiaphragmatic, gastric mucosa was present in all cases.

This is their summary:-

> "Thus the supradiaphragmatic dilatation observed during the act of swallowing in children without a stricture is to a great extent of gastric origin. This conclusion, it seems to us, must dispose of the idea that the temporary passage upwards into the thorax of the cardia, with more or less adjacent stomach, observed during deglutition is to be regarded as a hiatal hernia, and thus is a pathological condition. In our view, it is normal and a physiological phenomenon."

SIZE OF THE CARDIA

The variation in size of the cardia is well illustrated by Harrington's records (1928) of 500 digital examinations made during abdominal laparotomies. In sixty-five per cent the hiatus was tight round the oesophagus but in the remaining thirty-five per cent one finger or more could be inserted through the hiatus alongside the oesophagus. Of this thirty-five per cent, one finger was admitted in fifty-five per cent, two fingers in forty per cent and three fingers in five per cent.

THE CARDIAC SPHINCTER

The existence of a sphincter at the cardia is still a matter of great controversy. The recent intensive study of Lendrum (1937) and the work of Whillis (1931),

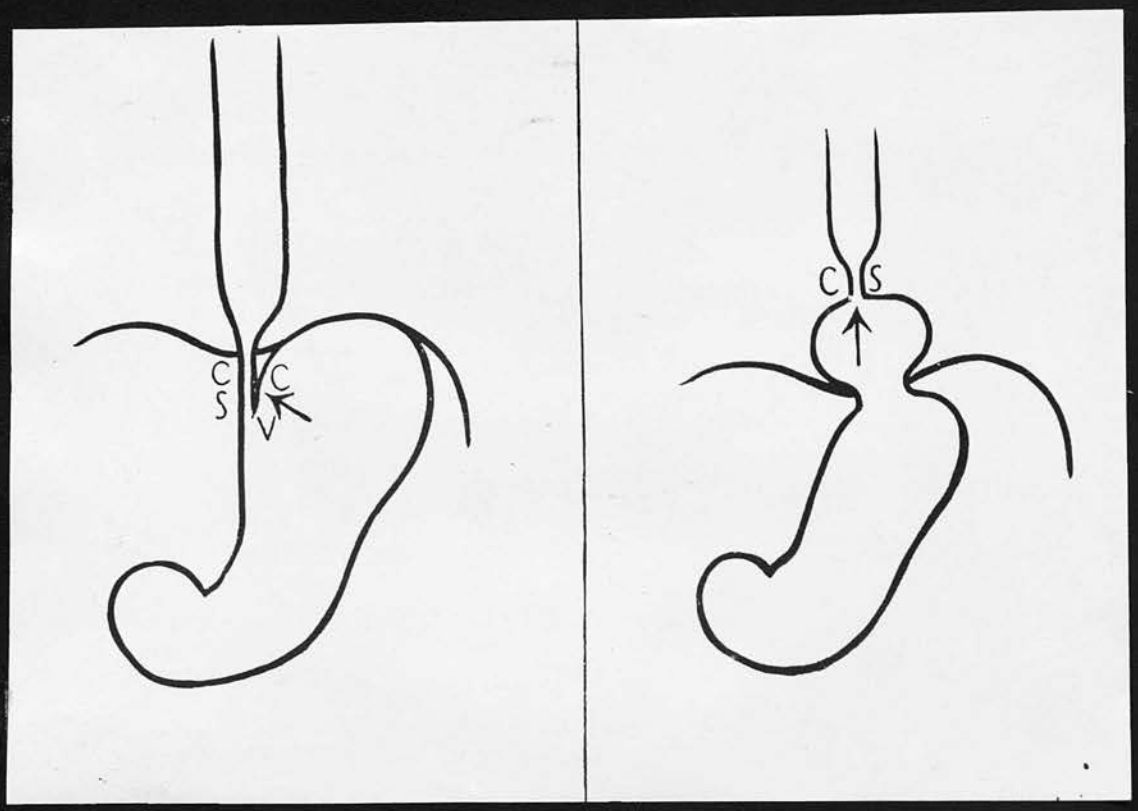
Jackson (1929) and others show that there is no true sphincter in the accepted morphological form. That is to say, there is no distinct band of circular muscle marked off from the adjoining muscle by connective tissue septa; there is no associated dilator muscle and there is no local thickening of circular muscle which persists after local spasm has been eliminated. The ∩-shaped band of muscle fibres described by Lendrum may, as he suggested, have a pinchcock action at the cardia.

Dick and Hurst, however, use Lendrum's observations to support their contention that the main resistance at the cardia is produced by a valve, formed by the angle of junction at the cardia. The left wall forms a flap which closes the exit when the intragastric pressure is raised (Fig.7).

They quote the ileo-caecal valve as another instance of the existence of a valve and sphincter. In congenital short oesophagus the cardia is situated at the top of the thoracic portion of the stomach and therefore the valvular mechanism is lost and there is no resistance to the regurgitation of the gastric contents when the presence of the thoracic pouch is sufficient to force the cardia (Fig. 8). Both authors ignore the influence exerted by the diaphragm.

Fig. 7

Fig. 8



cs, cardiac sphincter; cv, cardiac valve.

(Reproduced from Briggs and Hurst, Q.J.M. 1943 42: 109)

THE DIAPHRAGMATIC PINCHCOCK

This term was popularised by Chevalier Jackson (1929) who wrote that the true proximal sphincter of the stomach is the diaphragmatic pinchcock. Little notice, however, has been taken of the influence of the diaphragm on oesophageal emptying and the work of Whillis (1931) has almost escaped recognition in anatomical and physiological textbooks. The fundamental principles of Whillis's work are summarised as follows:-

"I. There is no histological evidence of a cardiac sphincter in Man.

"II. The cat shows a structure similar to that found in Man.

"III. In the cat and Man the oesophageal opening in the diaphragm is bounded in front by tendon and behind by the decussating fibres of the right crus. The intra-abdominal oesophagus is short.

"IV. The oesophagus of the rabbit contains three coats of striped muscle in its wall. The striped muscle is sharply demarcated from the plain muscle of the stomach. There is a well-marked thickening of the circular muscle in the cardiac region.

"V. In the rabbit and guinea-pig the intra-abdominal oesophagus is long and the opening in the diaphragm much larger than the oesophagus which traverses it.

"VI. Radiological examination shows:-

a. That in Man the diaphragm, when contracted constricts the lower end of the oesophagus, and

Fig. 9

A series of three radiographs which demonstrate the constricting action of the diaphragm during inspiration. The "hold-up" of barium is well seen in the middle frame.

Fig. 10

Illustrates regurgitation into the oesophagus when patient lay on right side and took a deep breath.

Fig. 11

Same patient. The radiograph shows vigorous wave of peristalsis emptying oesophagus.

"then when the diaphragm relaxes there is no further obstruction to the passage of barium emulsion into the stomach.

" b. The diaphragm has an action in the cat similar to that in Man. In the rabbit it has no obvious action on the oesophagus.

" c. Section of both vagus nerves produced no obstruction to the oesophagus in the cat.

" d. Section of both vagus nerves in the rabbit produced spasm of the cardiac region of the oesophagus, lasting for more than one hour."

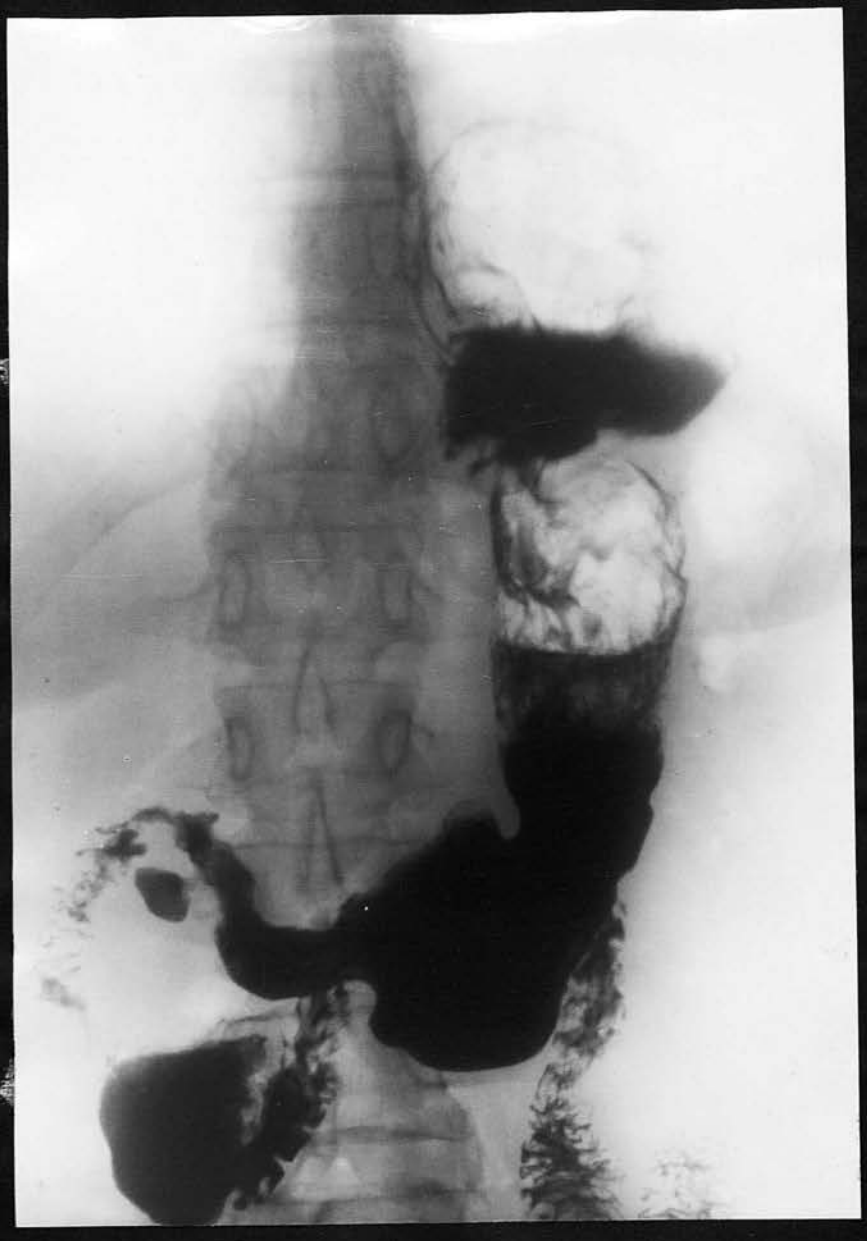
The writer has confirmed these observations in Man (Fig. 9) and it scarcely needs emphasising that any control which the diaphragm may exert on the oesophagus is lost in cases of hiatus herniae. This is probably the most important contributory cause of regurgitation. Fig. 10 illustrates the flooding of the oesophagus observed in a man of fifty who was given a barium meal in search for a cause of haematemesis. When he lay on his right side the barium regurgitated freely into the oesophagus during inspiration and then was emptied rapidly by a vigorous wave of peristalsis (Fig.11).

It would not be true, however, to say that apart from diaphragmatic control there is no check on regurgitation at the cardia. As Lendrum pointed out the anatomical structure in the last centimetre is different and there is just a localised narrowing which signifies the junction of

Fig. 12

TYPE I Hiatus Hernia Akerlund
Supine film. Gastric pouch filled. Oesophagus at apex of pouch. No reduction in erect position.

Fig. 13



Type 2 Hiatus Hernia.

Radiograph of a man aged 50 with multiple lesions (1) para-oesophageal hernia. The oesophagus is seen entering the stomach approximately at the oesophageal hiatus. (2) A duodenal ulcer (3) A duodenal diverticulum.

Fig. 14

Type 3 Hiatus Hernia. Apparently normal oesophagus and cardia when erect.

Fig. 15

Type 3 Hiatus Hernia. Same patient supine. Small pouch of gastric mucosa demonstrated at the hiatus.

oesophagus and stomach. The writer believes that the circular fibres may have some influence in producing this narrowing.

CLASSIFICATION OF HIATUS HERNIAE

Most of our knowledge of hiatus herniae has been based on the classical work of Akerlund (1926). He found these herniae to be six to seven times commoner than all other types of diaphragmatic herniae and classified them on an anatomical basis.

TYPE 1. - Oesophageal hiatus hernia with congenital shortening of the oesophagus (Fig.12).

TYPE 2. - Oesophageal hiatus hernia without congenital shortening of the oesophagus, in which the oesophagus does not form part of the hernia - a para-oesophageal hernia (Fig. 13).

TYPE 3. - Oesophageal hiatus hernia in which the oesophagus is not shortened but the distal part of the oesophagus forms part of the hernial contents. This is the sliding type of hernia (Figs. 14 and 15).

Akerlund's observation that elderly people, particularly of the sthenic build show marked mobility of the cardia has been amply confirmed. Schatzski's work published in 1932 has considerable bearing on this observation. He took an unselected group of thirty

patients between the ages of sixty-five and eighty-three and by special radiological examination combined with abdominal pressure he showed hiatus herniae in 50 per cent. This figure was increased to 73.3 per cent when the colon was distended with air to increase intra-abdominal pressure. He made several interesting observations after analysing sixty-six cases which he had found within two years. The average age was sixty-eight and there was no sex preponderance. When a hernia was found in middle-aged patients, they were of the sthenic build, but after the age of sixty-five all types were equally affected. Certain causal factors were described: loss of fat in the hiatus, loss of elasticity in the peri-oesophageal connective tissues and a stretching of the muscle bundles forming the hiatus. Stress was laid on the increase in the intra-abdominal pressure, particularly when cases were found in the middle age groups. Schatzski observed in one case the development of a hiatus hernia in association with the growth of enormous secondary deposits of cancer in the liver. He considered these herniae to be acquired, on account of their frequency, their preponderance in old age, and the effect of increasing intra-abdominal pressure. He likened the hernia to the development of inguinal or femoral herniae in the aged, due to loss of elasticity

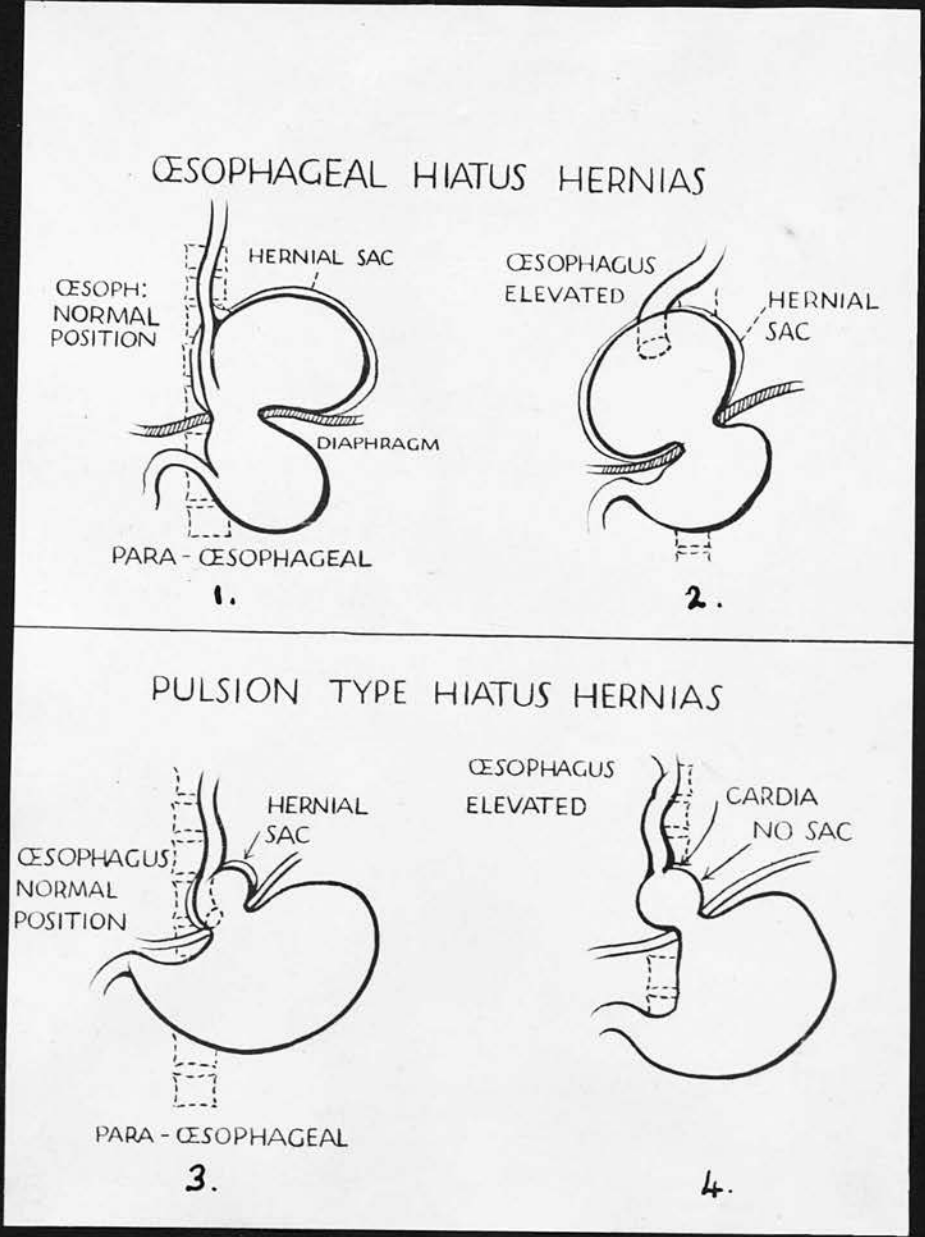
with senile tissue changes , the mechanical factor in the two groups acting in different directions, as the erect position produces one group, the recumbent position the other.

Rigler and Eneboe (1935) found eighteen per cent of a series of pregnant women to have a hiatus hernia during the last trimester. The frequency of heartburn in the later stages of pregnancy is attributed by Evans and Bonslay (1940) to the increased intra-abdominal pressure and the development of hiatus hernia.

Harrington in 1943 published his revised classification of hiatus herniae, drawn up on a clinical basis, and his recently added types are in keeping with the observations of Schatzski (1932), Allison et al. (1943). Harrington states that the hiatus hernia is the most common type of diaphragmatic hernia, a point on which there is general agreement. He considers it to be a slowly progressive projection of the stomach, usually alone, into the thoracic cavity. He believes that it is best to classify all true herniae through the oesophageal hiatus under one term: "Oesophageal hiatus diaphragmatic hernia." He describes four main types (Fig. 16):-

TYPE (1) - The oesophagus is of normal length and is not projected above the diaphragm, but a portion of the

Fig. 16



1. Oesophageal hiatus hernia with herniation of the cardiac end of the stomach through the oesophageal hiatus. Oesophagus in normal position (para-oesophageal hernia.)

2. Oesophageal hiatus hernia in which there is herniation of the greater portion of the stomach through an enlarged oesophageal hiatus into the posterior mediastinum and associated elevation of the oesophagus.
Harrington Am.J.Surg.1943 492189

3. Slight protrusion of the cardia of the stomach through the hiatus with slight elevation of the lower part of the oesophagus to the upper margin of the oesophageal hiatus. This is a pulsion type of herni with incompetent hiatus.

4. Protrusion of cardiac end of the stomach through the oesophageal hiatus and marked elevation of the oesophagus into the posterior mediastinum; incompetent hiatus.

Fig. 17

Type (1) Harrington's classification. Para-oesophageal hernia. Oesophagus of normal length. About a quarter of the cardiac end of the stomach lies above the diaphragm.

Fig. 18

Type (2) Harrington's Classification. Gastric pouch projecting to the left side.

Fig. 19

Type (2) Harrington's Classification. Gastric pouch projecting to the right side.

stomach herniates into the posterior mediastinum beside the oesophagus. These herniae are usually small, rarely involving more than a quarter to a half of the cardiac end of the stomach, which is contained in the hernial sac (Fig.17).

TYPE (2) - The oesophagus is of normal length but its lower end is elevated above the level of the diaphragm and the herniated stomach is in the posterior mediastinum. The size of the hernia is generally larger than in the case of Type (1) and may contain the whole stomach with a portion of omentum and colon within the sac. The hernia fills the mediastinum and usually projects to the left (Figs. 18 and 19).

The difference between these types - (1) and (2) - is one of degree rather than a fundamental difference in origin.

The two remaining types are recent additions and may be considered 'pulsion' types. In these cases a small portion of the cardiac end of the stomach projects through the oesophageal hiatus. This may be due to atrophy of the elastic fibres of the diaphragmatico-oesophageal membrane, together with relaxation of the circular muscle surrounding the hiatus, with a resultant incompetent hiatus.

They occur commonly in elderly persons and in many instances are accidental findings.

TYPE (3) - Herniae of this type closely simulate the para-oesophageal type of hernia except that they are small. A definite sac is beginning to form. The oesophagus is only slightly elevated and the abdominal portion is level with the superior border of the diaphragm. (Fig.20).

TYPE (4) - The hiatus is incompetent, the cardiac end of the stomach protrudes like a funnel above the diaphragm. The oesophagus is elevated and enters at the apex of the protruded portion of the stomach. There is no hernial sac (Fig.21).

CONGENITAL SHORT OESOPHAGUS

Odgers (quoted by Dick and Hurst) stated that congenital shortening of the oesophagus occurs when the oesophagus fails to elongate with sufficient rapidity to keep pace with the growth in length of the embryo. A portion of the stomach is consequently pulled up from below the septum transversum to form a true hernia. The elongation of the oesophagus is most active in the 5 - 10 mm. embryo during the fourth and fifth weeks and it is therefore likely that the hernia dates from this very early stage of development.

The older embryological explanation was that the oesophagus fails to grow to a normal length when the

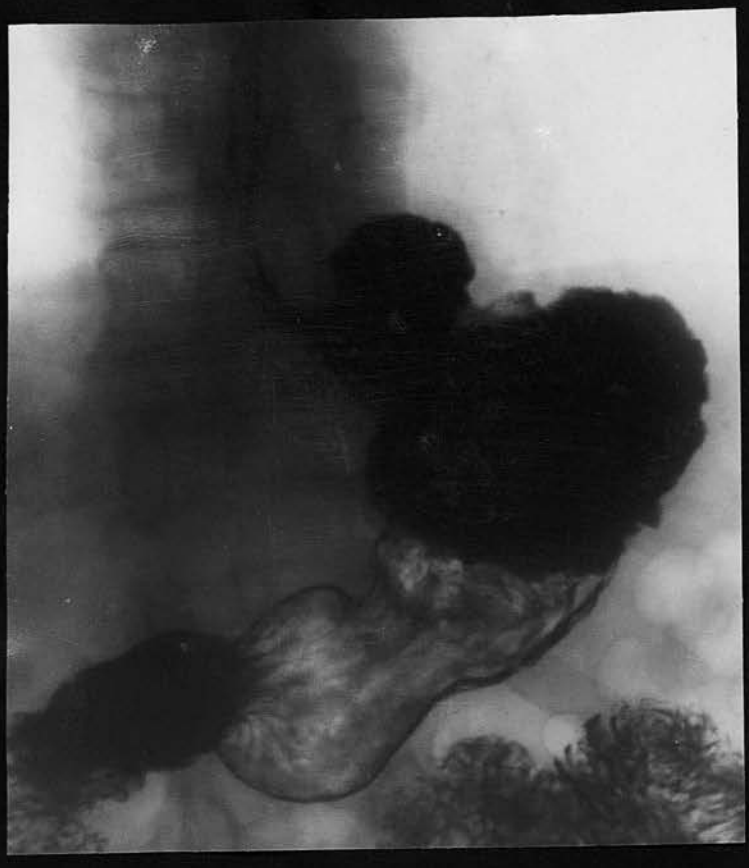


Fig. 20

Type (3) Harrington's Classification.
Hernia closely simulates para-oesophageal type only it is small.

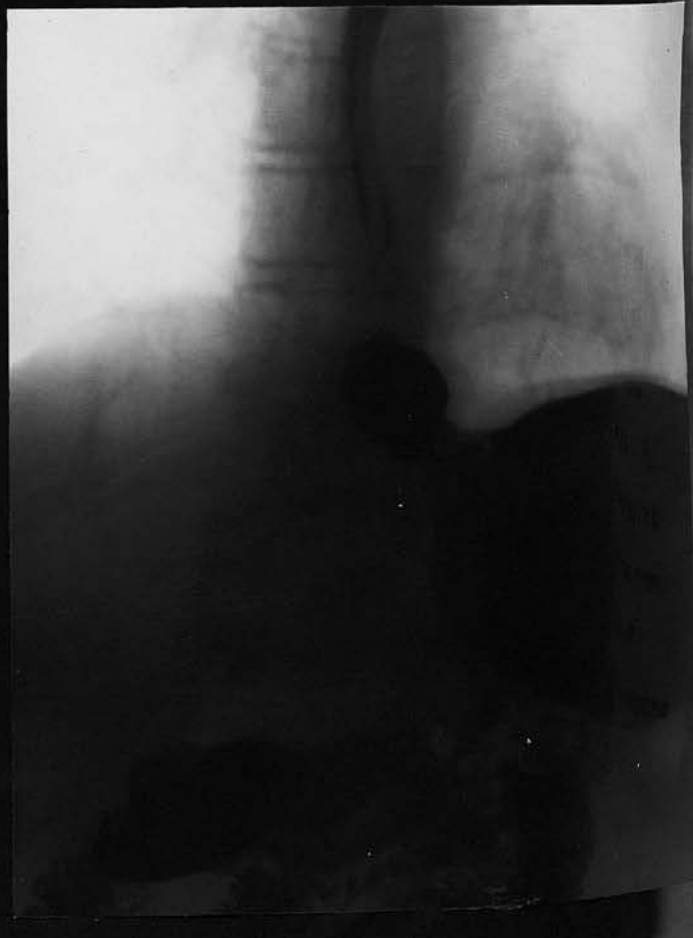
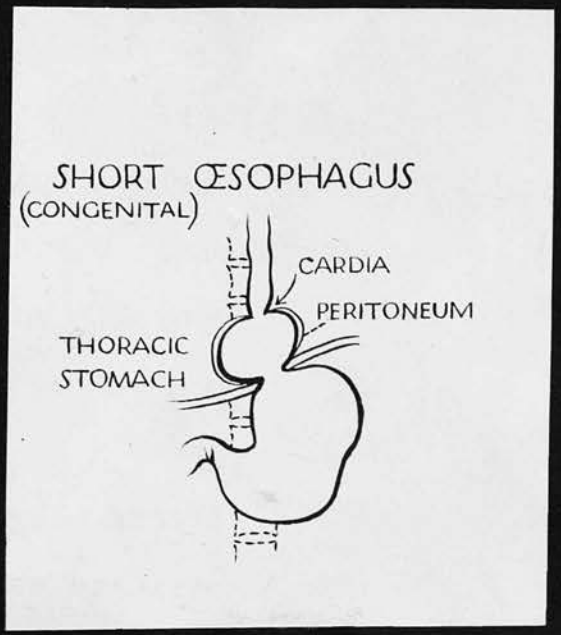


Fig. 21

Type (4) Harrington's Classification.
Cardiac end of stomach protrudes through diaphragm. Oesophagus enters apex of pouch.

Fig. 22



Congenital short oesophagus with partial thoracic stomach. (Harrington)

stomach and septum transversum are displaced downwards by the rapidly enlarging heart and lungs. A portion of the stomach is retained within the thorax and is termed a partial thoracic stomach. Strictly speaking, according to Findlay and Brown Kelly (1931), it is not a hernia, as that part of the stomach has never been within the abdomen but it is generally accepted as a hernia and was classified by Akerlund as Type 1. hiatus hernia (Fig. 22).

In Harrington's experience true congenital shortening is very rare. In eleven patients who presented radiological evidence of congenital short oesophagus only four were found at operation to be true cases of shortening. In four others reduction of the hernia was easily performed and in the remaining three there was only very slight shortening with herniation of the cardiac end of the stomach.

CONGENITAL OR ACQUIRED SHORTENING OF THE OESOPHAGUS

To recapitulate the various views advanced it will be recalled that Briggs, Dick and Hurst in their original publication (1939) stated that in all their cases the shortening of the oesophagus was congenital and ulceration occurred mostly in people beyond middle age. Jackson (1929) had previously found no abnormality in

the oesophagus other than ulceration and almost all his patients were young adults. Allison, Johnstone and Royce (1943) drew attention to these contradictions and pointed out that while in some cases the shortening may be congenital there was abundant evidence that in the majority it might be acquired. They based their argument on the following observations - many of which have already been described in more detail.

ACQUIRED SHORTENING

All their cases of proved peptic ulceration have occurred in middle-aged or elderly people. If the shortening was congenital why does ulceration not develop at an early age, especially when the incidence of peptic ulceration of the stomach and duodenum is greatest in the third and fourth decades.

With advancement in radiological diagnosis the recognition of hiatus herniae has been fairly common and it has been shown to occur most frequently in elderly, stout people. Striking figures from the Mayo Clinic are given by Harrington (1943) who records that between 1908 and 1925 only thirty cases of diaphragmatic hernia were diagnosed but between 1926 and 1941, sixteen instead of eighteen years, there were 600 cases. This he ascribes to better radiological technique rather than an increase in incidence.

Fig. 23

Fig. 24

These radiographs illustrate small pulsion herniae - Type (4) - frequently encountered in routine work.

Fig. 25

Fig. 26

Fig. 27

Sliding hiatus hernia - not found at post-mortem.

The effect of increasing intra-abdominal pressure has already been discussed in reference to the work of Schatzski (1932), Rigler and Eneboe (1935). Many cases of pulsion hernia have been encountered by the present author in routine hospital work and examples are illustrated by Figs. 23 - 26.

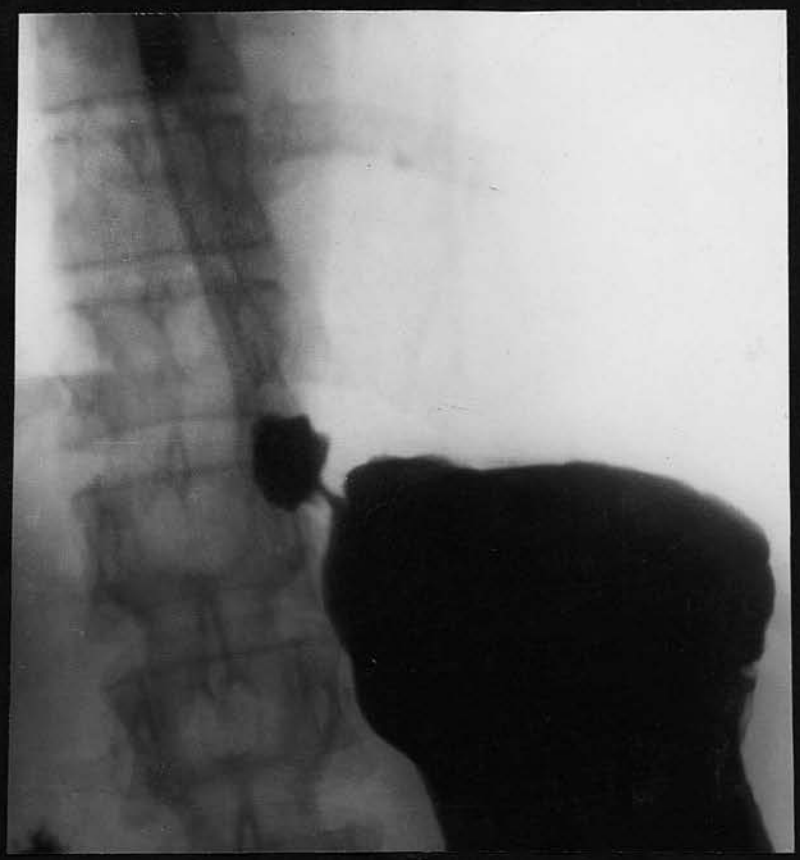
From these it seems fair to assume that the cardia, which may well have some normal mobility, tends to become herniated with increasing years and weight. One might justifiably ask why these herniae have not been found more frequently at autopsies. It is well established that they are nearly always missed and Fig. 27 shows an excellent example. This patient was reported as having a hiatus hernia and yet, with the full knowledge of its existence, the pathologists could not demonstrate it.

Lyall (1937) has shown that chronic ulceration of the oesophagus may lead to intensive fibrosis and although there is a pronounced tendency to constrict the lumen, shortening in a longitudinal direction may occur in a manner analagous to the purse bag stomach. The longitudinal muscle constantly exerts a tension towards a reduction in length of the oesophagus. This is well

Fig. 28

Illustrates the attachment of the oesophagus to the aorta and the resulting distortion caused by unfolding of the aorta. This tends to elevate the cardia. A small gastric pouch is seen above the diaphragm.

Fig. 29



Visceroptotic patient supine position. Small pouch suspected but unproved. Note the angulation at the gastric fundus directed towards or through the hiatus.

illustrated in the contraction which occurs immediately after its surgical removal. In another case, following total gastrectomy, the jejunal anastomosis was found to have been dragged up into the thorax. Further, one constantly sees in elderly patients with some arterio-sclerotic changes in the aorta a distortion of the oesophagus owing to its attachment to the unfolded arch (Fig. 28). This must give rise to quite an appreciable tension on the lower half of the oesophagus.

Again, in elderly people whose build is not hypersthenic there is a tendency for the abdominal viscera and diaphragm to sag downwards. This would increase the strain on the oesophagus and tend to drag the cardia towards or through the hiatus. (Fig. 29).

The theory of acquired shortening is now supported by Harrington (1943) who stresses the rarity of the congenital short oesophagus and considers that ulceration and occasionally malignant neoplasm of the oesophagus have caused cicatricial contraction of the lower end which draws the cardia above the diaphragm.

CONGENITAL SHORTENING

Dick and Hurst (1943) assert that the condition is always congenital. They quote the case described by Haroen and Gerlings (1934) which occurred in a three-year

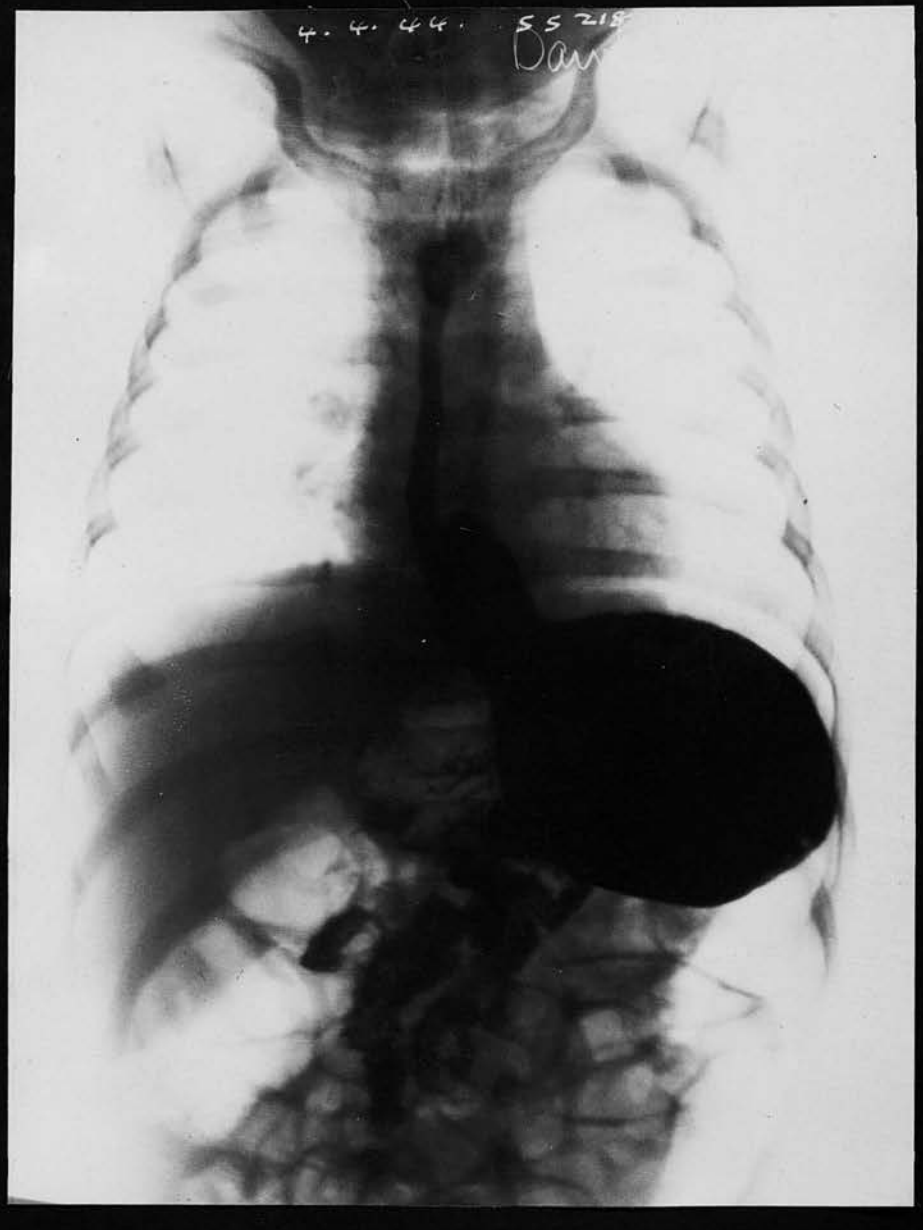
old child whose symptoms began when she was a week old, so that the short oesophagus was almost certainly congenital. On the other hand they describe one of their own cases, a man of seventy-eight, who developed a severe fibrous stricture of the oesophagus and gave a history suggestive of ulceration for six years. No shortening of the oesophagus was found and they consider that if fibrosis was the predominant factor in its production then it should have been present in this case.

Dick and Hurst also hold that when a case occurs in which no herniated gastric pouch is found then the ulceration is due to isolated heterotopic gastric mucosa. They point out that in their series very little fibrous stenosis was encountered. Dysphagia was generally absent and when present it was due to spasm as it disappeared when the ulcer healed.

DISCUSSION ON THE CONGENITAL THEORY

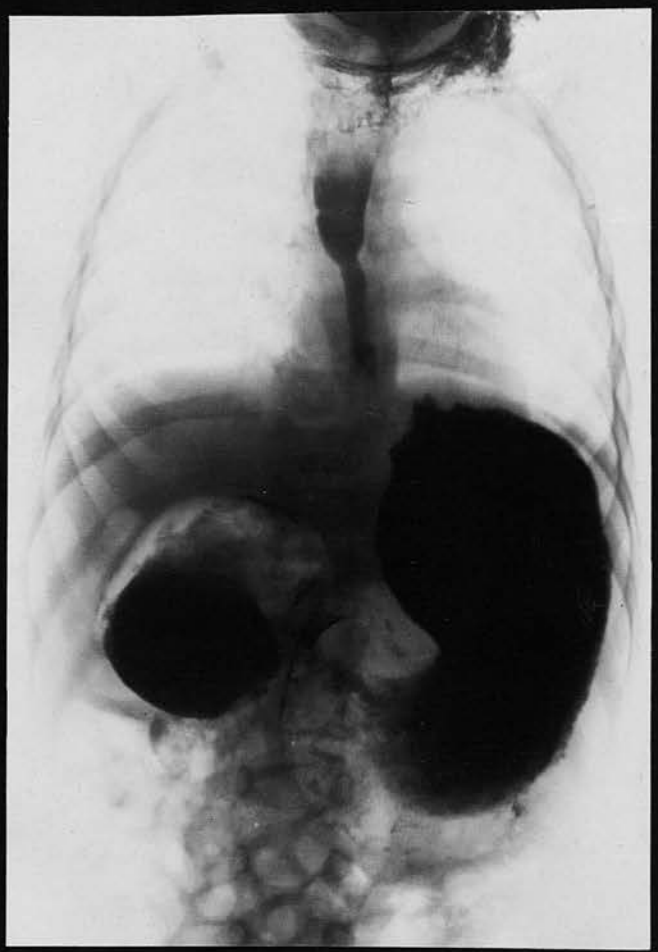
That all these cases are of congenital origin is borne out neither by Harrington's observations (1943) nor by the work of Findlay and Brown Kelly (1931). If there is a physiological hernia of the cardia through the hiatus then it becomes increasingly difficult to decide if the oesophageal shortening is congenital or acquired. Even when the condition is associated with

Fig. 30



Baby, four weeks old, showing short oesophagus with stenosis and partial thoracic stomach.

Fig. 31



One-year-old child with duodenal atresia. Vomiting occurred daily. Oesophagus shows narrowing and commencing shortening.

stenosis in infants it is not possible to be sure on this point for Kelly (1939) described ulceration with fibrosis and shortening of the oesophagus in the very young.

He says:

> "In some instances this chronic inflammation remains strictly localised but in others it appears to spread widely up and down the tube, the spreading inflammatory cellular reaction preceding the fibrosis."

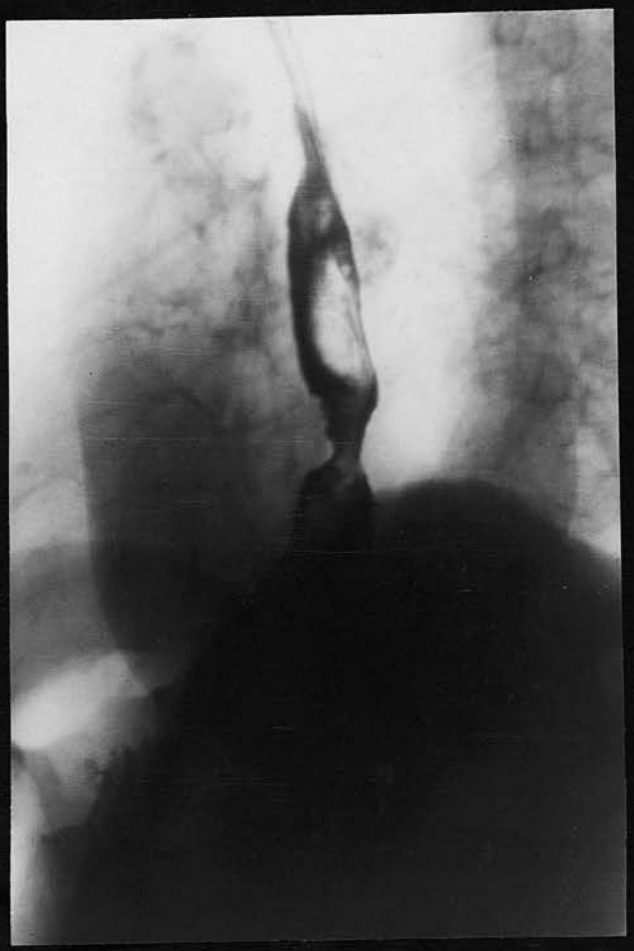
The true congenital type is demonstrated by the radiograph of a child four weeks old who had had intermittent vomiting since birth and in whose vomit the doctor noticed streaks of blood at the age of three days (Fig. 30). A typical short oesophagus with stenosis and herniation of the stomach is clearly shown. Fig. 31 is a radiograph presenting a somewhat similar appearance but the subject was a one-year old child who had vomited daily since birth. Her weight was only 7 lbs. The anatomical cause of the vomiting was a congenital atresia of the second part of the duodenum and the constant regurgitation had given rise to chronic inflammatory changes at the lower end of the oesophagus. The wall shows marked thickening and the level of the cardia was so high that commencing herniation was suspected.

SUMMARY AND COMMENT

There appears to be sufficient evidence that most cases of peptic ulcer of the oesophagus are associated with acquired shortening of the oesophagus and herniation of the stomach. In some instances, however, the shortening appears to be congenital (Fig. 32).

The sequence of events in a typical case might well be as follows: an active young man, of hypersthenic build, with hyperacidity develops a duodenal ulcer. This may eventually heal with or without surgical aid. In middle age he puts on weight and develops a pulsion type of hiatus hernia. As a result of this, acid regurgitation into the oesophagus occurs frequently and leads to ulceration just above the cardia. If symptoms arise they may be mistaken for a recurrence of the duodenal ulceration. The oesophageal lesion may alternately heal and break down leading to much fibrosis, and the oesophagus both shortens and narrows so that the hernia becomes irreducible and the oesophageal lumen obstructed. Dysphagia then becomes the predominant symptom.

Fig. 32



The peptic ulcer seen left is situated at a much lower level than that on the right. The latter is so high that it must be due to a congenital shortening.

CLINICAL FEATURES

Peptic ulcers have been found in patients of all ages, but they are commonest in adults, especially over fifty. In this series the predominating features were suggestive of oesophageal obstruction which it must be admitted indicate a relatively late stage in the disease. Chevalier Jackson (1929), who saw the largest series in early stages, wrote that symptoms may be completely absent or they may resemble those produced by a gastric ulcer. The diagnosis is further complicated by the fact that gastric and duodenal ulceration may frequently co-exist with oesophageal ulcers.

The symptoms Jackson described in his patients were pain behind the lower end of the sternum and high up in the epigastrium, sometimes going through to the back between the shoulder blades, coming on one half-hour or longer after food is eaten and relieved by alkalies, heartburn, vomiting, haematemesis or melaena. In half the cases there was complaint of pain over a small, easily localised area as a bolus of food passed over the ulcer. The ulcer in these cases was old and peptic in character, evidently having originated long before the subjective sensation of pain. Dysphagia was late and

associated, of course, with stenosis.

Dick and Hurst (1943) give a detailed description of the pain which, they find, has certain characteristics. Pain, burning or smarting and often described by patients as heartburn, is almost invariably present. It is felt whilst eating or drinking and at first occurs only when a hard substance, perhaps insufficiently chewed, is swallowed, or it may be brought on by strong thermal or chemical irritants. Later it accompanies any meal but bland fluids may be taken without discomfort. The pain lasts only a few minutes and is relieved by alkalies, but later it becomes more prolonged. The intensity varies and it can be severe enough to make the patient frightened to eat.

Pain is also felt an hour or two after meals if the patient bends forwards. It frequently returns on lying down. It may wake him in the early hours of the morning. It is often accompanied by the regurgitation of very acid clear fluid. Sitting or standing up may bring relief and the pain may be prevented by tilting the bed or using extra pillows at night.

Dick and Hurst suggest that the pain felt while eating may be a reflex protective spasm of the oesophagus immediately above the ulcer. The pain on leaning forwards or lying down is due to irritation of the ulcer by acid

regurgitation. This suggestion does not conform with the accepted statement that peptic ulcers in the stomach and duodenum are insensitive to irritants including hydrochloric acid.

In the early stages the symptoms occur in attacks separated by periods of freedom but may continue with varying severity for years. Later stenosis may develop and the dysphagia becomes more pronounced, with the regurgitation of food and saliva, often mixed with streaks of blood. They found occult blood present in all cases. Three cases had haematemesis and one of these had melaena.

Allison et al. (1943) reached the conclusion that peptic ulcer with short oesophagus gives the following clinical picture:-

1. The patient is usually middle-aged or elderly.

2. The pain is felt behind the lower end of the sternum and sometimes strikes between the shoulders. It comes on one-half to one hour after food. It may be severe or trivial. It is relieved by alkalies and increased by bending forwards.

3. There is often pain at the lower end of the sternum immediately after swallowing which may persist

until the bolus passes or until induced retching brings it back. Relief after regurgitation is pronounced.

4. The duration of pain varies from a few months to several years. The co-existence of gastric or duodenal ulcer and biliary disease complicate the picture. Of the seventeen cases one had a gastro-enterostomy for duodenal ulcer and subsequently developed a jejunal ulcer, one suffered a severe haematemesis from a duodenal ulcer shortly before his oesophageal ulcer started and one had been operated on for peptic ulceration but the site was not known. Two patients had had cholecystectomies and in one gall-stones were seen on the films.

5. Vomiting may occur although this may in fact be regurgitation of oesophageal contents.

6. Haematemesis occurred in four cases. It may be severe and even fatal.

7. Dysphagia is the outstanding symptom. Its onset may be relatively sudden. It may vary from day to day. The patient may learn to obtain relief by tickling his throat to cause regurgitation, or taking alkalies. He avoids coarse foods, strong chemical or thermal irritants. In this way he may keep relatively free from trouble and does not lose weight.

RADIOLOGICAL EXAMINATION

In the technique used in the routine examination, the patient is first screened in the erect position while barium emulsion is swallowed. By rotating the patient and screening during different respiratory phases a complete survey of the oesophagus is made and films are taken to record abnormal findings. The right and left oblique positions are generally the most useful. This is followed by the swallowing of a teaspoonful of thick barium paste, which sticks longer in the oesophagus and more clearly defines slight spasms or narrowings. The paste is not used when there is any marked obstruction.

The same procedure is then carried out with the patient lying horizontally or with the head a little lowered. Moderately powerful manual compression of the abdomen may be used but the more drastic method of colonic insufflation devised by Schatzski (1932) has not been employed. It is well recognised that pressure is best applied at the end of respiration when the hiatus is relaxed. Rotation into the right oblique plane was found to be most useful as the gastric pouch filled readily in this position.

Radiologically the observations must be separated into two groups. The first is directed towards identifying the abnormality in the lower end of the oesophagus and the

second towards determing whether or not there is a gastric pouch herniating through the diaphragm. The lesion at the lower end of the oesophagus may reveal itself by a spasm, above which there is a little dilatation. Below the spasm one may find a small ulcer niche in a short narrowed area and then the channel widens out showing typical gastric folds. Early cases probably show no niche, for the ulcer is little more than an erosion. In one case the wall was found to be uneven, semi-rigid and to vary a little during the passage of barium. In this case the fibrosis was predominant but oesophagoscopy did reveal a small ulcer (Fig.32a).

The cases in this series were mostly advanced and certain characteristic features were found. The upper oesophagus was widened to a varying degree, but not so extensively as in achalasia. Some thickening of the walls was noted but no abnormal peristalsis. The oesophagus tapered down rather abruptly into a zone of stenosis, which was generally high, about 6 to 12 cms. above the hiatus. Its length ranged from 3.5 to 6 cms. Near the upper end of the segment there might be a well-defined deep ulcer but more often the crater was shallow and impossible to distinguish from irregularity due to spasm or fibrosis. (Fig.33). Differentiation from a neoplasm was more often than not impossible. Leucoplakia, which caused in two cases small

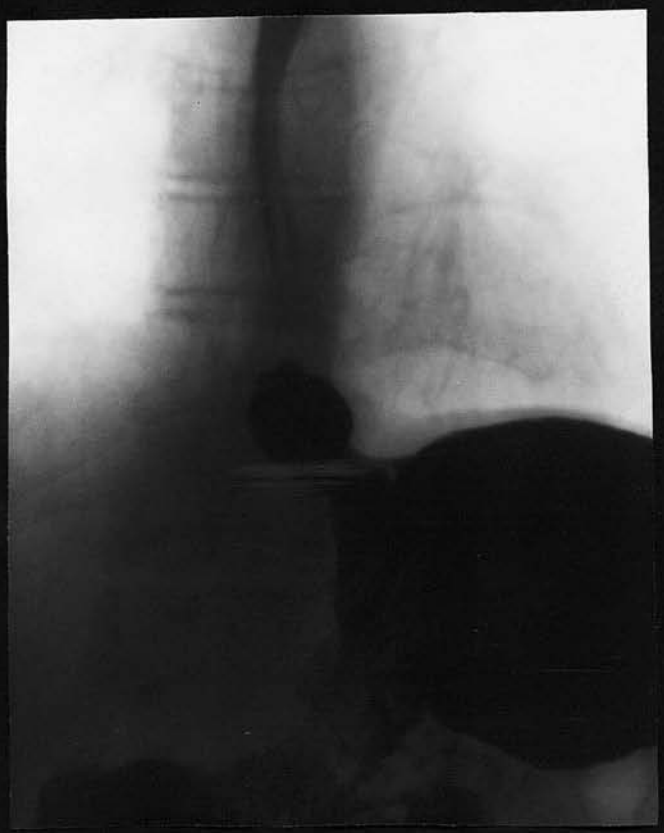
Fig. 33

Radiograph showing characteristic features as described on p. 35.

Fig. 34

Fig. 36

Fig. 35



Top left: erect. Lower end of oesophagus appears normal. Lower left: supine. Small gastric pouch demonstrated. Pulsion type of hernia. Top right: Radiograph showing sudden regurgitation into oesophagus when lying supine and on the right side.

Fig. 37

Erect. Apparently normal oesophagus.

Fig. 38

Supine, Same case. Coarse gastric folds in nipple-like projection into hiatus.

Fig. 39

Fig. 40

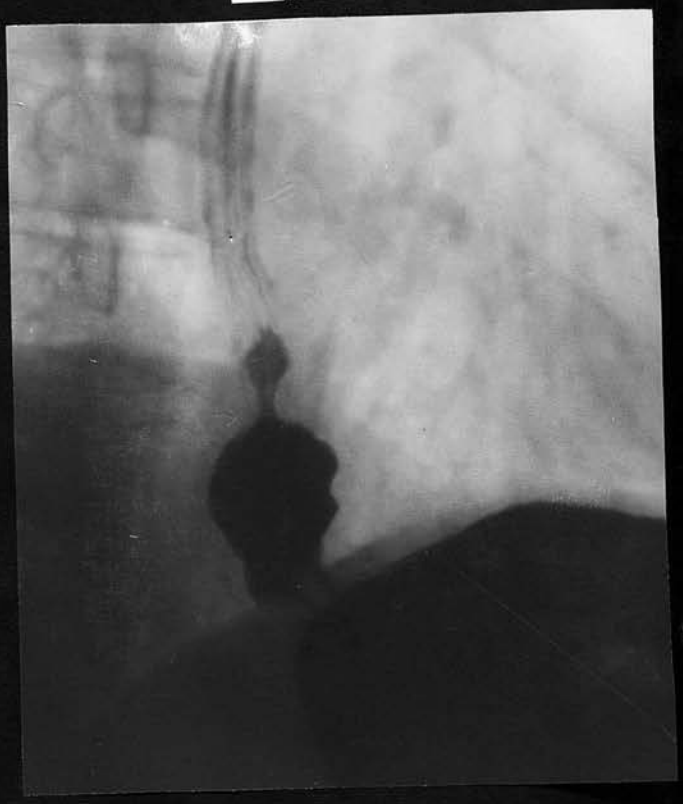
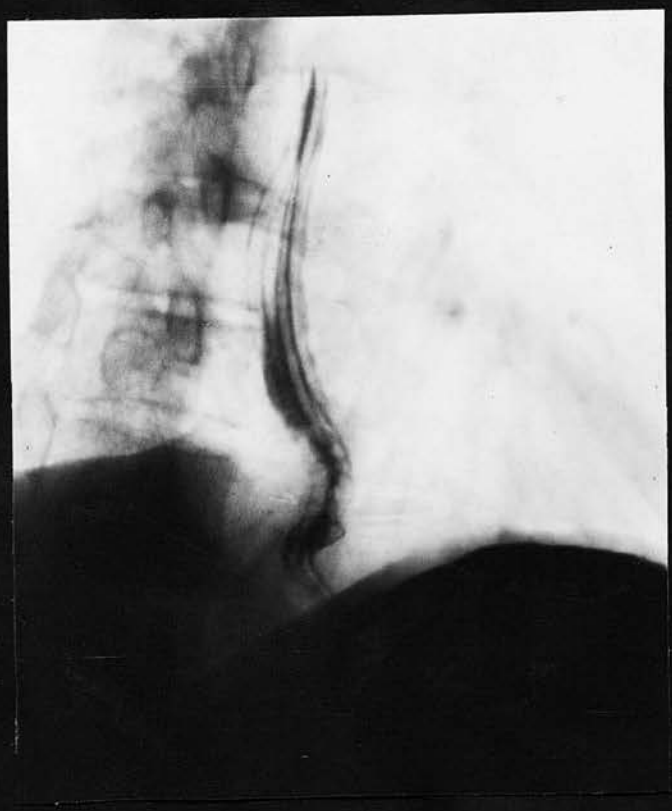


Fig. 41



Three radiographs of the same patient showing (top left) coarsening of mucosal folds with slight varicosity: (top right) small gastric pouch filled with barium: (Lower left) another phase when mucosa has oesophageal appearance. A slight spasm at cardia is constant - marked with arrow.

Fig. 42

Left radiograph shows pseudo-pouch in the epiphrenic portion of the oesophagus. Note conical apex.

Right radiograph shows normal oesophageal mucosa.

The above radiographs show in contrast a gastric pouch, definitely proved by the clip method.

filling defects, gave rise to more confusion.

The exit from the stenotic region was generally abrupt and opened out into a wide channel, varying in size according to the position of the patient and showing the typical gastric mucosal pattern.

The diagnosis and classification of hiatus herniae is based essentially on the radiological evidence and depends to a large extent on determining the position of the cardia. The main distinguishing feature is the change from oesophageal to gastric mucosa, the former being characterised by fine and the latter by coarse, sometimes granular, longitudinal ridges, which are met by varicose folds running at right angles from the greater curvature (Fig.34-41). Owing to varying phases in the depth of the gastric mucosal relief it may be difficult to distinguish the change, but one is helped by noting the position of a constant spasm or narrowing which represents the cardia. This again may be confused by a spasm at the epi-cardia, and not infrequently a bolus of barium is locked just above the hiatal portion so that it resembles a small gastric pouch. One differential point is that in the pseudo-pouch the shape of the bolus is olivary with a conical apex. Fig. 42 shows a pseudo-pouch in a young adult. The mucosal folds are a little coarse but there is neither varicosity nor irregularity.

Having established the position of the cardia and filled the gastric fundus to its capacity by the various manoeuvres described, one determines the category into which the hernia falls. In the true sliding type the hernia will reduce itself in the erect position and this can be observed while screening.

ENDOSCOPIC EXAMINATION

Allison records the following:-

"The diagnosis must finally rest on an accurate oesophagoscopic picture with a biopsy. The oesophagus above the stricture is usually slightly or moderately dilated. It may contain an excess of cloudy fluid, but the mucous membrane is normal except at its lower end. At a level, which in our patients has varied between 26 and 34 cms. from the alveolar margin, there is narrowing of the lumen. The mucous membrane immediately above the stricture is usually bluish-white with fine dark lines upon it, which gives the appearance of crocodile skin. This area oozes blood readily if touched with the oesophagoscope. Shallow ulcers may be seen in this area, but it is not usual for the chronic ulcer to be visible until the stricture has been dilated. During dilatation free bleeding may occur from injury to granulation tissue, but this has not been dangerous in our cases. Unless a dry field is obtained it may be difficult to make a diagnosis. The easiest mistake is to see the upper end of the pouting gastric rugae and assume it to be growth. More careful inspection, however, will reveal the ulcerated area immediately above this. The upper end of the ulcer is usually irregular, with bright red areas of granulation tissue interspersed with thin white processes of epithelium. We have only taken minute biopsies and this has not given rise to any complication. It is often difficult to be certain within one or two millimetres where the biopsy forceps are in contact when the tissue is taken. This is particularly true if the oesophageal wall is freely movable or if the gastric

"mucosa projects upwards. Some variation in the histological picture of these biopsies is therefore to be expected and it may be necessary to examine the patient more than once before malignant disease can be excluded."

DIFFERENTIAL DIAGNOSIS

The combination of a stenotic segment with an ulcer crater in the lower oesophagus and a hiatus hernia is strong presumptive evidence of peptic ulceration.

As the patients are elderly the most important differential diagnosis is carcinoma of the lower end of the oesophagus or of the gastric fundus with an oesophageal extension. It must be recognised that in the cases with marked obstruction any irregularity in the zone of stenosis should be regarded as strongly suggestive of a neoplasm or leucoplakia and the diagnosis is only made by endoscopy and biopsy. In these cases one may fail to get sufficient barium into the stomach to flood the fundus and fill a gastric hiatal pouch, and therefore the combination of lesions is not found.

The height of the stenotic zone above the diaphragm may be a useful guide in the differentiation because cancer generally attacks the oesophagus at the bifurcation of the trachea or in the ampulla cardiaca whereas peptic ulceration is usually found midway between the situations.

Fig. 43

Left oblique, supine. Carcinoma of lower oesophagus with partial thoracic stomach. Stenotic area showed much irregularity.

Right oblique, erect. Same patient.

Fig. 44

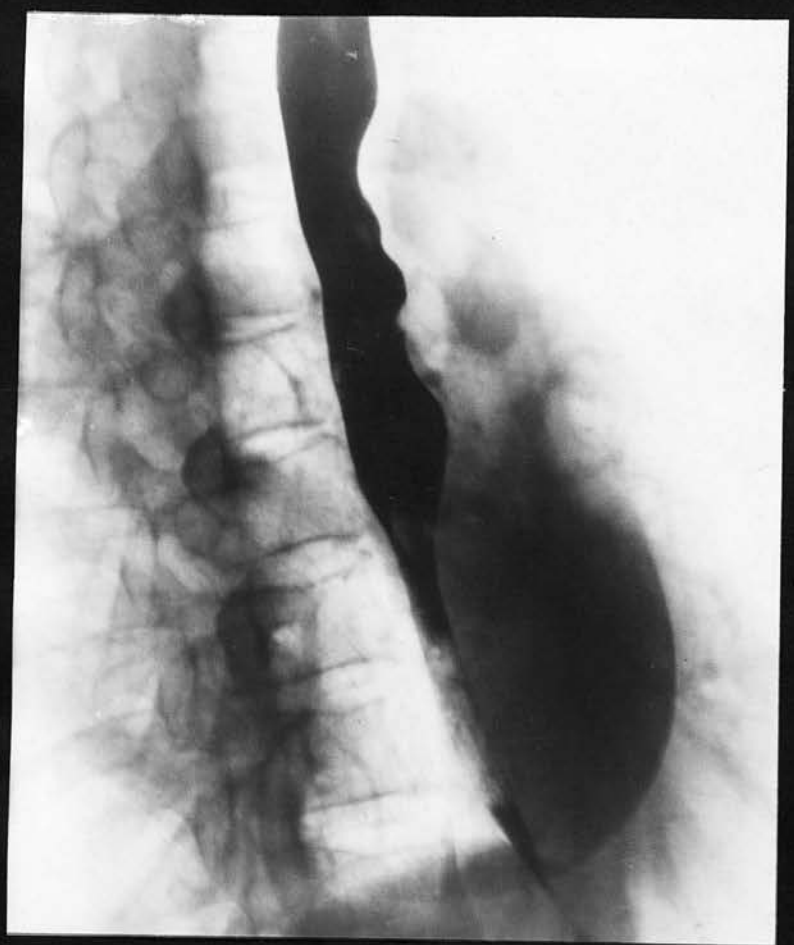
Supine. A case of carcinoma of the lower oesophagus with a partial thoracic stomach. Note the deep ulcer with undermined edges. No marked fibrosis. Obstruction only moderate.

Same patient erect. Hernia almost reduced.

Fig. 45

A case of carcinoma of the fundus of the stomach. The filling defect is seen outlined by the gas bubble.

Fig. 46

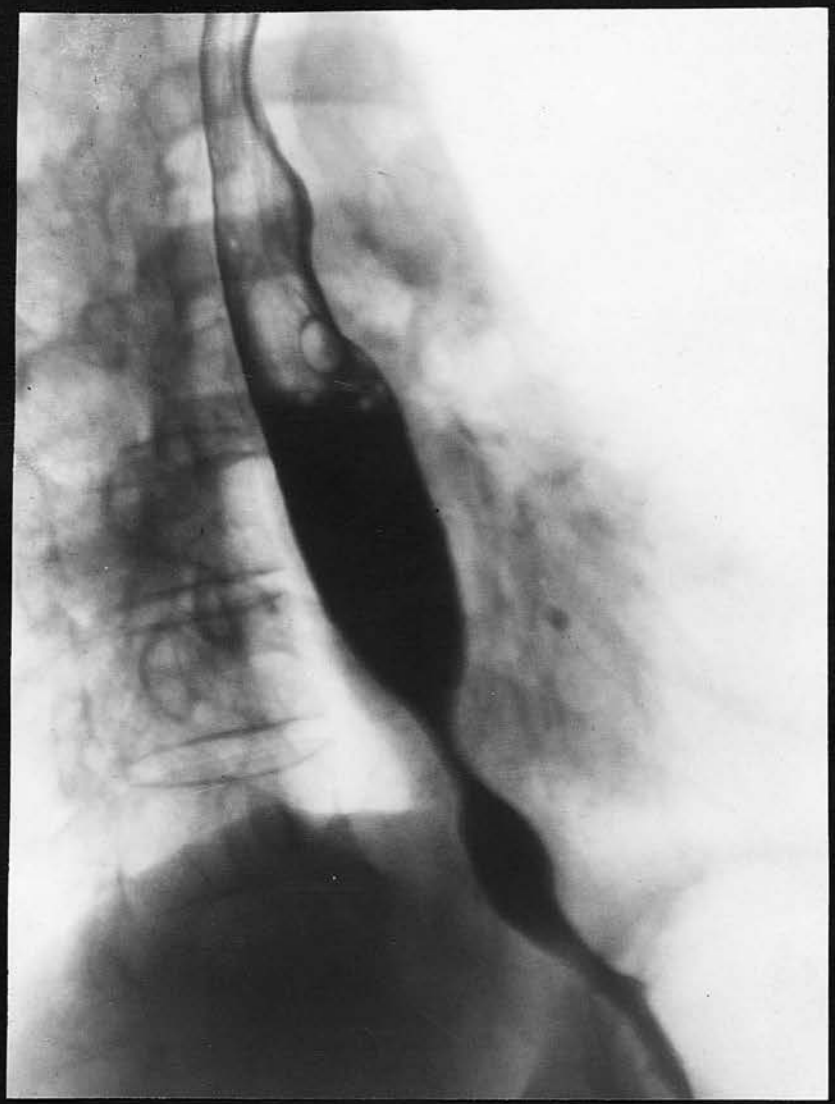


Radiograph of chronic stricture in the lower third of the oesophagus due to swallowing a chemical irritant 20 years previously. Complete obstruction had occurred due to the unsuspected swallowing of a plum stone which is seen at the neck of the stricture.

Fig. 47

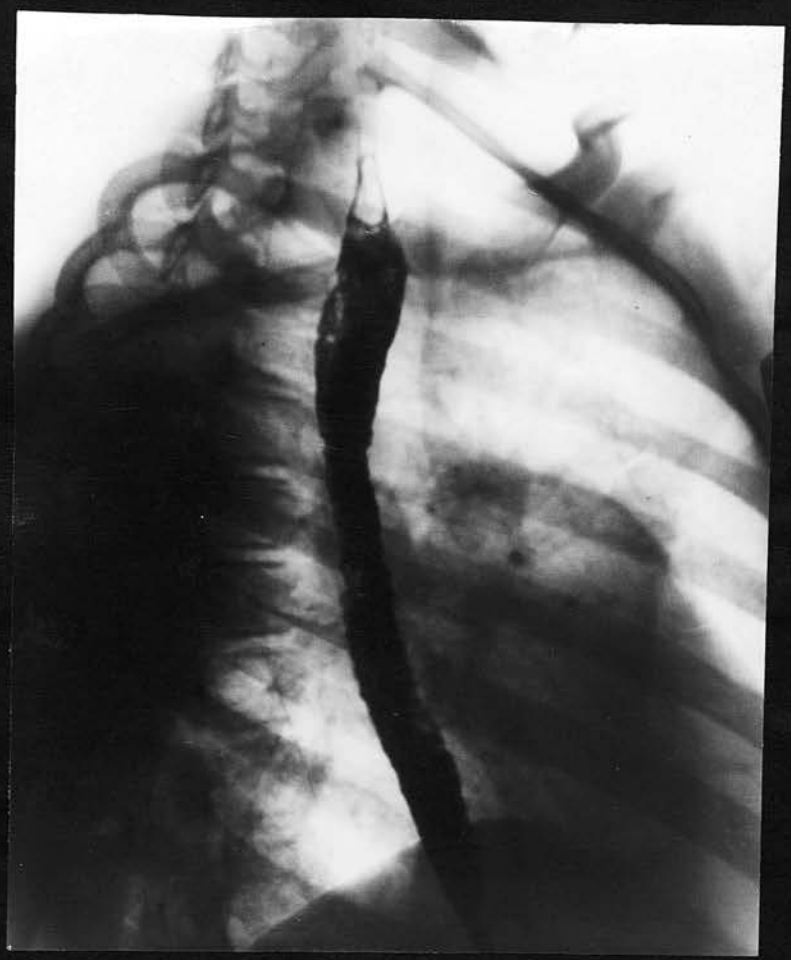
Congenital stenosis in mid-third of oesophagus with shortening of the oesophagus. The change to gastric folds is clearly seen above the diaphragm.

Fig. 48



Illustrates a case of peptic ulceration (see Case 15) after treatment with a stenotic area indistinguishable radiologically from a simple stricture.

Fig. 49



Acute oesophagitis fourteen days after swallowing caustic soda. There is extensive irregularity due to ulcerated mucosa and spasm.

Fig. 50

Semi-erect. Irregular stenosis in oesophagus just above cardia. Coarse rugae of gastric pouch seen above the diaphragm.

Fig. 51

Same case, supine. Impossible to exclude ulceration or malignancy. Endoscopy showed oesophagitis only.

Some cases of cancer of the lower oesophagus combined with a partial thoracic stomach have been found and examples are seen in Figs. 43 and 44. The oesophageal irregularity leaves little doubt about the diagnosis but the ulcer in Fig. 44 might be simple. The columnar-celled carcinoma arising from the ampulla cardiaca may extend up the oesophagus but generally it is accompanied by a spread into the stomach which is often thrown into relief by the gas bubble. A careful inspection of this part of the stomach must be made (Fig. 45).

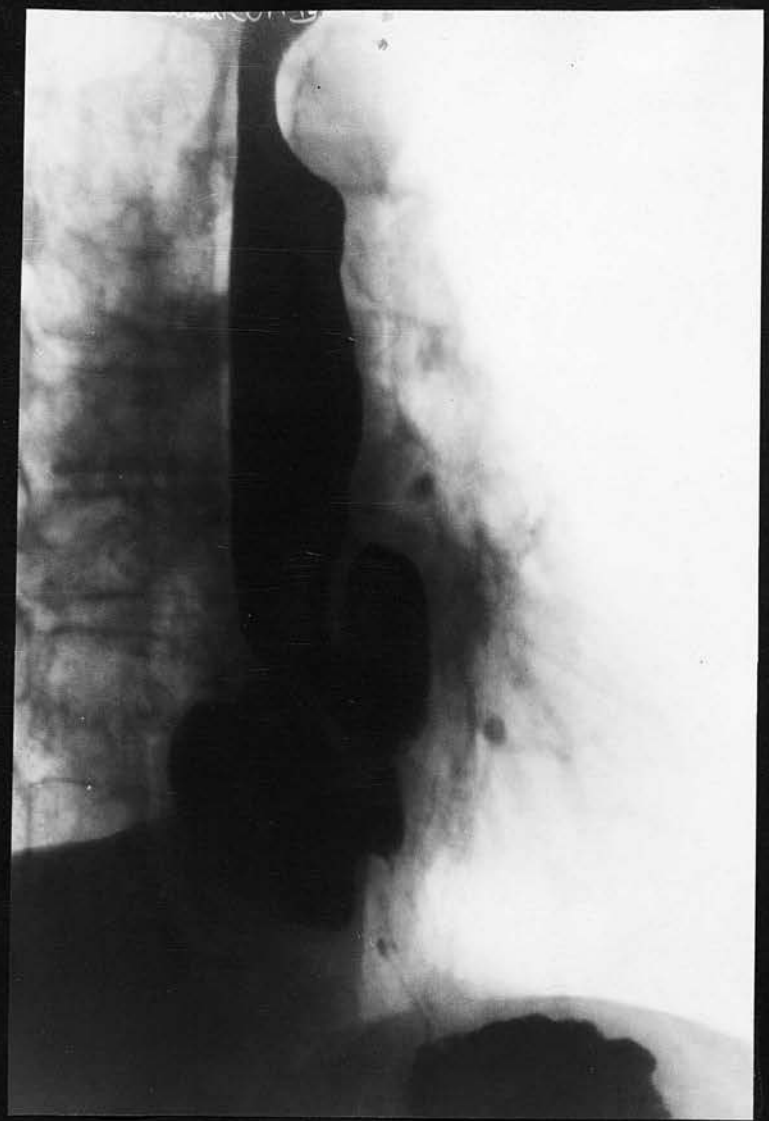
Other conditions which must be considered are simple stricture, oesophageal diverticulum and oesophagitis.

In the simple stricture there is generally a history of trauma, either thermal or chemical, and a progressive dysphagia. The stenosis is tapering, smooth and no crater is observed. A gastric hiatal pouch is probably not present (Figs. 46 and 47). However, some cases of ulceration which show improvement following treatment would be difficult to distinguish from simple stricture (Fig. 48).

Acute oesophagitis is generally found in patients who have recently swallowed some caustic and the spastic state of the lower part of the oesophagus is characteristic (Fig. 49).

Chronic oesophagitis may be quite indistinguishable radiologically and the differentiation depends entirely on finding an ulcer by oesophagoscopy (Figs. 50 and 51).

Fig. 52



Two large epiphrenic diverticula found in a man aged 54, suffering from haematemesis.

Fig. 53

Small projection above the cardia ? diverticulum, ? early type (3) hernia. There is a duodenal diverticulum which favours the former but gastric folds can be traced into the small sac.

illustrate a case where the diagnosis of peptic ulcer and partial thoracic stomach was made but no ulceration was found. Biopsies showed only a sub-mucous, round-celled infiltration.

Most writers stress the epicardiac diverticulum of the oesophagus as a possible source of error but Fig.52 shows the characteristic features of the latter and these in no way resemble peptic ulceration. (Compare, however, Fig. 53).

Tuberculous ulceration need only be mentioned for the lung infiltration would be noted on screening.

TREATMENT

The treatment of the simple oesophageal ulcer is largely based on appreciating that only under certain conditions does acid regurgitation occur into the oesophagus and that the oesophagus is at rest except during deglutition. A strict régime of rest is ordered but the patient should sit up as much as possible during the day and sleep with several pillows or have the head raised at night. He should be given a few large feeds as opposed to the small, frequent feeds of a Sippy diet. Foods should be restricted to fluids or semi-solids and anything very hot or cold must be avoided. Dick and Hurst recommend a pint of milk, which need not be citrated, four times a day. Some of the milk can be replaced by junket, arrowroot, Benger's, Horlick's, custard or thick white vegetable soup, but the last should not be very hot or salty.

About four ounces of water should be drunk five minutes after each feed to wash away any traces of food which might stick to the mucosa at the lower end of the oesophagus. An ounce of strained orange or tomato juice is given with two of the feeds and 100 mgms. of ascorbic acid, dissolved in milk, should be given daily.

Atropine sulphate gr. 1/100, dissolved in one drachm of water should be given one quarter of an hour before each feed to reduce the tendency to spasm. The dose should be increased by 10 mins. daily until signs of intolerance or overdose appear. The dose is then reduced to that of the previous day. A tablespoonful of olive oil should be drunk immediately before each feed; if this is unobtainable one or two teaspoonfuls of cod liver oil act equally well.

The strict regime is continued until the patient has been symptom-free for at least a fortnight, three consecutive stools have contained no occult blood and the radiographs show no crater.

Dick and Hurst suggest that if no diaphragmatic hernia is present then it is assumed that heterotopic islets exist in the oesophagus. In these cases treatment is given on the lines of a duodenal or gastric ulcer. The free use of bismuth lozenges is recommended.

In this series the dysphagia was relieved dramatically by oesophagoscopic dilatation but this may require repeating at varying intervals. Some of the ulcers were painted with twenty per cent silver nitrate but this failed to give any striking results.

In three cases the improvement was only temporary even after repeated dilatation so more drastic surgical procedures were advised. One patient had a gastrostomy, another a jejunostomy and in both there was a striking improvement. The jejunostomy was allowed to close in six months. The third patient refused a gastrostomy and a year later was in moderately good health but still complaining of dysphagia. It is possible that in some of the intractable cases a surgical excision of the affected segment may be the operation of choice. (See Appendix B.)

With symptomatic improvement changes are generally seen during radiological examination. The crater may become smaller or disappear, the spasm and dilatation above it become less. The walls seem less rigid and more expansile.

In no case, however, was there complete recovery even when the ulcer healed. A varying amount of narrowing remained at the site and the thoracic stomach was unchanged. On endoscopy signs of persistent ulceration were generally present.

The association of hiatus hernia and hypochromic anaemia has recently gained prominence in medical literature. The descriptions suggest that bleeding occurs in the chronically congested gastric pouches formed in the para-oesophageal types of hernia. When anaemia exists iron therapy is indicated and the response is said to be very good.

AFTER-CARE

When the ulcer has healed, the patients should be put on a post-ulcer régime, similar to that provided for gastric and duodenal ulcer. The head of the bed should be raised permanently and they should always take a glass of plain water five minutes after a meal.

Slow eating, efficient mastication and adequate dentures are necessary.

PROGNOSIS

The disease is serious and complete cure does not readily occur. With dietatic measures and dilatation when necessary it should be possible to keep a patient living comfortably and in sufficiently good health to carry out normal duties. It should be borne in mind that haemorrhage or perforation may occur at any time and prove fatal.

A P P E N D I X A

CASE REPORTS

Summarised reports of eighteen cases are now presented. Seventeen of these are proved peptic ulcers of the oesophagus with partial thoracic stomach. The eighteenth is unproved but provides useful material for the general discussion on diagnostic difficulties.

The first ten cases have already been published (Allison et al. 1943) and notes of the remainder have been extracted from the records of the Thoracic Surgery Department of the General Infirmary at Leeds.

All these cases are under the care of Mr. P.R. Allison and have been radiographed by the author.

CASE 1 - C.S. was a woman aged 53 years.

HISTORY - The patient was first seen 6th December, 1939. In October 1938 she had complained of solid food being held up just below the manubrium sterni. When obstruction occurred she would obtain relief by induced regurgitation. There were occasional remissions and sometimes two days passed without trouble. The

Fig. 54

CASE 1 - Supine position, showing small gastric pouch filled with barium and mass of infiltrated tissue around ulcer projecting into it giving a "uterine cervix" defect Some mucosal swelling is evident just above the stenosis.

Fig. 55

CASE 1 - Erect position, showing stenosis with flat ulcer crater on postero-lateral border. Coarse longitudinal rugae are seen above the diaphragm. No marked dilatation is noted above the stenosis.

Fig. 56

CASE 1 showing changes after dilatation. The ulcer crater is no longer visible and the stenotic area is wider.

dysphagia was at times associated with pain behind the lower end of the sternum. She felt a pain in the same place when she lay down and this was relieved by induced vomiting. The dysphagia became constant in July 1939 and since that time she could swallow only very soft or fluid foods. There was no loss of weight and no history of indigestion before October 1938.

EXAMINATION - Radiography in December 1939 showed narrowing of the lower end of the oesophagus with a small herniation of the stomach into the mediastinum. The oesophagus was short. Irregular peristalsis was observed and an ulcer crater was suspected on the postero-lateral wall of the narrowed segment (Figs. 54 and 55). Endoscopy was performed 12th December, 1939. During the administration of the local anaesthetic a small quantity of coffee-ground fluid was regurgitated. At a point 26 cm.. from the alveolar margin the mucous membrane of the oesophagus showed a patchy white area like leucoplakia and immediately below this, on the anterior wall, was a shallow ulcer 2 cm. by 2.5 cm. from which a biopsy was taken. Dr. Stanbury reported that one small fragment showed stratified squamous epithelium in which the superficial layer of cells showed considerable variation in

size, shape and staining reaction with some parakeratosis. Other fragments were composed of rather dense granulation tissue infiltrated by plasma cells and lymphocytes. The appearances were those of a simple ulcer with surrounding leucoplakia.

PROGRESS - Two years later the patient was swallowing well, suffering no pain but complained of regurgitations occurring when she lay down (Fig. 56).

CASE 2 - The patient, R.W., was a 55-year old woman.

HISTORY - Thirty-four years ago, immediately after the birth of her first child, the patient suffered from "Bad indigestion and terrible heartburn." This lasted for three years and during that time she was constantly sick in the morning. She frequently brought up bilious fluid but never any blood. For thirty years she complained "on and off" of a burning pain under the lower end of the sternum which struck between the shoulders. It would come on at any time and frequently awakened her at night. It was always worse when she was lying down, so that if she rested in the afternoon the pain came on, but if she walked about it did not. It was relieved by alkalies. For two years she had complained that food

would stick at the lower end of the sternum and cause pain but when it passed on she felt some relief. She would obtain relief by self-induced regurgitation and then might eat the rest of the meal without difficulty. The dysphagia was intermittent. There was no loss of weight. She was seen by us in July 1940 when radiography showed no delay of fluid barium swallowed when the patient was in the erect position. In the supine position a short oesophagus with herniation of the stomach was shown and the last 6 cm. of the oesophagus were irregularly contracted. Above this, the oesophagus was dilated and showed very little active peristalsis. An ulcer was seen 1.5 cm. above the cardio-oesophageal junction (Fig. 57). In the erect position there was apparent reversion to the normal.

Endoscopy showed a constriction of the lower end of the oesophagus 30 cm. from the alveolar margin. The face of the stricture was smooth and a bluish-white colour. A No. 30 bougie was easily passed through it, the walls giving way readily before only slight pressure. After dilatation, moderate oozing of blood occurred and when this had been stopped an ulcer 2.5 cm. by 1.5 cm. was seen on the posterior wall of the oesophagus. The

Fig. 57

CASE 2 - Supine film showing irregular stenotic area with ulcer crater on right lateral border. Slight dilatation above. Gastric pouch well-outlined.

CASE 2 - Supine film after dilatation. Ulcer crater still visible but stenotic area shows improvement.

upper margin of this area was irregular with finger-like processes extending up into the white area and the lower margin was concealed by the pouting folds of gastric mucosa. A biopsy from the pale area showed squamous epithelium with leucoplakia; one from the ulcer consisted of inflammatory tissue covered by a purulent necrotic layer and one from below the ulcer was reported as gastric mucosa showing considerable oedema and infiltration by chronic inflammatory cells.

CASE 3 - The patient was A.J., a man aged 68 years.

HISTORY - The patient complained of a feeling of pressure behind the lower end of the sternum for three months. It came on suddenly when he was eating and then only intermittently. It would start behind the lower end of the sternum and rise up until the pain became so intense that he had to induce regurgitation for relief. His food might then go down more easily. The difficulty was first experienced with solid foods but later fluids were sometimes held up. There had been no loss of weight. There was no definite previous history of gastric trouble except that in 1933 he had had a cholecystectomy for what had been described as gastric catarrh. The trouble had been present for three or four years and was completely relieved by operation.

EXAMINATION - He was first seen in January 1940 when radiological examination was done. There was no obstruction to fluid barium but narrowing of the oesophagus was seen to start about 9.5 cm. above the diaphragm and there was a holdup of semisolid barium at this point. The lumen was narrowed like the stem of a wine-glass and at the neck there was a small filling defect. Below the narrowed region the lumen expanded into what was seen to be herniated stomach (Fig.58).

Endoscopy was done 5th March, 1940. At a point 30 cm. below the alveolar margin there was an area on the posterior wall of the oesophagus about 2.5 cm. long with a mammillated surface. It looked like a localised elevated patch of leucoplakia. The surface was bluish-white and it was thought that it might even have been a collection of varicose veins. A biopsy was taken from it and oozing was free. The oesophagoscope was carefully passed beyond it and an ulcerated bleeding area was found. Details of this were not easily made out because of the blood, but a second biopsy was taken. Below this area the folds of gastric mucosa were seen. Dr. Stanbury reported that the first biopsy consisted of a small piece of stratified squamous epithelium showing prolongation of the rete pegs and in places a thickening of the malpighian layer. Many cells of the superficial layer

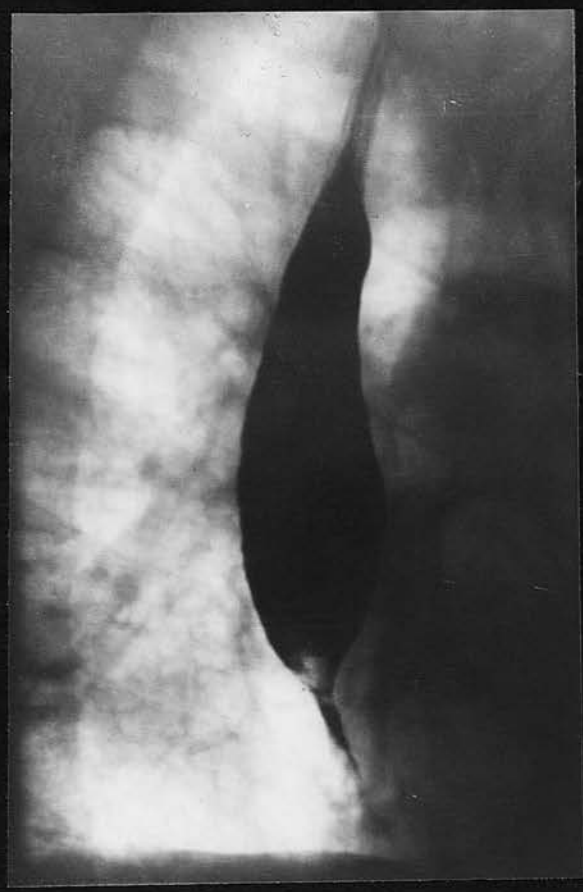


Fig. 58

CASE 3 - Erect position, showing oesophagus dilated and tapering gradually to the stenosis. A small defect is seen at the entrance; crater not seen.

CASE 3 - Supine position, right oblique; suspected ulcer can be seen on the posterior wall. The gastric pouch is partially filled.

were vacuolated. A number of smaller fragments showed hyperkeratinization. Between and beneath the rete pegs there was a sparse infiltration of chronic inflammatory cells, chiefly lymphocytes. The diagnosis was leucoplakia. The second biopsy consisted of a small piece of tissue clothed by columnar epithelium containing numerous goblet cells. The subepithelial tissues were loose, oedematous, and heavily infiltrated by chronic inflammatory cells, chiefly plasma cells. The tissue had the appearance of gastric mucosa with well-marked chronic gastritis. It seems likely that the biopsy forceps had gone beyond the ulcer in this case.

PROGRESS - This patient was seen fifteen months later and was very well.

CASE 4 - A.M. a woman, 69 years of age.

HISTORY - The patient had been complaining of difficulty in swallowing for six or seven years. It was not associated with pain and she had practically no indigestion. She felt that the food was held up at varying points throughout the gullet. While the difficulty had been gradually increasing it was typically intermittent and variable in degree so that an entire day might pass

without trouble and then she might feel worse than ever. She frequently regurgitated froth and sometimes food, but had never vomited blood. She had not lost weight.

EXAMINATION - Radiographs taken in May 1940 showed a short oesophagus with partial herniation of the stomach through the diaphragm. Barium was held up 4.5 cm. above the cardia where the lumen was narrowed to about 4 mm. for a distance of 1.5 cm. On the anterior aspect of this narrow zone was a prominence suggesting a crater. There was a little dilatation of the oesophagus above the stricture.(Fig.59). On 7th May, 1940 an oesophagoscope was passed. At a point 28 cm. below the alveolar margin, the oesophagus was concentrically narrowed to a diameter of about 5 mm. The mucous membrane above this was healthy. Bougies up to size No. 28 were passed through the stricture and were felt to be gripped quite firmly. Some oozing of blood occurred so that a clear view of the inside of the stricture was not obtained but a biopsy was taken from it. Dr. Stanbury reported that the second specimen consisted of a piece of gastric mucosa showing chronic gastritis. The first specimen consisted of a small piece of gastric mucosa which was the seat of a chronic gastritis and a small amount of necrotic material

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Fig. 59

CASE 4 - Supine position. An ulcer crater is seen on the anterior wall 3 cms. above a moderately large gastric pouch. Note also facetted gall-stones.

Fig. 60

CASE 4 - Erect position; semi-solid barium, three months after Fig.59 . The ulcer has nearly healed.

infiltrated by large numbers of polymorphonuclear neutrophiles and moderate numbers of lymphocytes, plasma cells, and eosinophilic leucocytes. The finding is important inasmuch as the ulcer from which this biopsy was taken was at least 2 cm. above the gastric mucous membrane. It must, therefore, have been an area of heterotopic gastric mucosa. When reviewed after three months of medical treatment the patient was much better. A radiographic examination showed marked improvement but it appeared that the ulcer was not completely healed (Fig. 60).

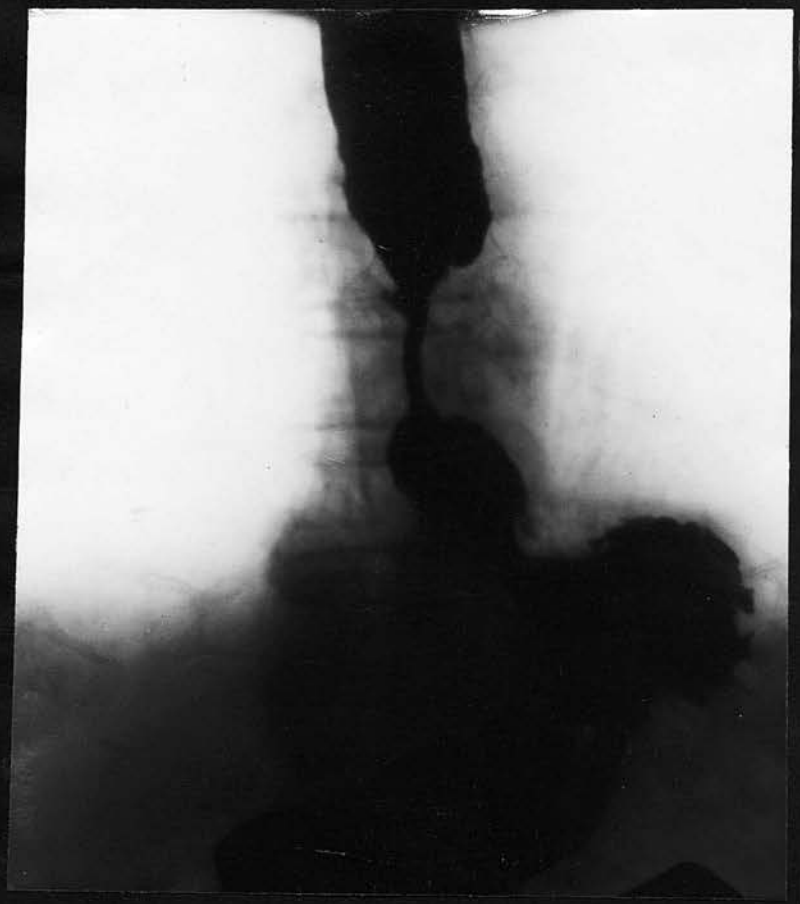
CASE 5 - The patient was W.S., a male, aged 80 years.

HISTORY - When first seen in June 1940, he complained that in January he had noticed slight difficulty in swallowing solid food. This gradually increased and in April 1940 he was admitted to another hospital for abdominal pain and vomiting. On that occasion he vomited three pints of blood. Since that time his only complaint was dysphagia. Fluids passed down and he could swallow soft bread and butter but anything more solid seemed to stick at the top of the sternum. He complained of a heavy feeling behind the sternum but no pain. He lost thirty-five pounds in weight in six months. Since having his gall bladder removed in 1911, he frequently

Fig. 61

CASE 5 - Erect, left oblique; air distending the gastric pouch below the ulcer shows longitudinal striae continuous with gastric rugae. The dome of the diaphragm appears to be dragged upwards by the oesophagus.

Fig. 62



CASE 5 - Supine position showing the gastric pouch filled; the extent of the stenosis and ulcer crater is well illustrated.

complained of pain in the epigastrium about two hours after meals. It sometimes made him vomit but was always relieved by bismuth mixture.

EXAMINATION - Radiography showed a constriction of the oesophagus 9 cm. above the diaphragm. The stricture was 3.5 cm. long and opened below into a dilated portion which was part of the stomach drawn up into the mediastinum. At the upper end of the stricture on the right anterolateral aspect was an ulcer crater 0.5 cm. to 1 cm. deep. Above the stenosis, the oesophagus was moderately dilated (Figs. 61 and 62). Endoscopy was performed 6th June, 1940. At a point 34 cm. from the alveolar margin the oesophagus was constricted and on the right anterolateral wall was an inflamed pit about 1 cm. in diameter. It was impossible to see into this, but an occasional bubble was seen to come out of it. The stricture was dilated and two biopsies were taken, one from the left wall just above the stricture, which showed stratified squamous epithelium with a shallow area of ulceration, much infiltration by inflammatory cells, and extensive fibrosis of the musculature, a second from the ulcer itself which showed both stratified squamous and gastric type epithelium intensely inflamed with active chronic ulceration. All his symptoms disappeared on

treatment, but a radiograph taken 7th October, 1940 showed a crater still present. One year later the patient still complained of intermittent dysphagia.

CASE 6 - This patient D.S.S., was a male aged 64 years.

HISTORY - He had complained of dysphagia for about four months. The difficulty was associated with pain behind the lower end of the sternum while food was passing down. He had had treatment for a jejunal ulcer so that it was not possible to say if the oesophageal ulcer had been causing the abdominal symptoms. The pain gradually subsided but the dysphagia increased so that solids and semisolids could no longer be taken. The dysphagia varied in degree so that at one time he would decide to have something done for relief and at another would be persuaded that he was improving. On one occasion when a little fish stuck in the gullet and caused severe pain, he brought up three or four ounces of bright red blood. He complained of excessive salivation but this in reality was the accumulation of saliva in the gullet and throat. Finally, the obstruction became complete and the patient was admitted to the nursing home in a very distressed condition. He apparently had not lost weight. He had had a gastroenterostomy for duodenal ulcer twenty-seven years before and

had been well until the development of the jejunal ulcer.

EXAMINATION - Radiological examination elsewhere had shown a narrowing of the oesophagus about 4 cm. long. The lower end of this was fairly smooth and there was some dilatation of the oesophagus above. Immediately below the narrowed part the tube was dilated and this portion was found to be stomach herniated through the diaphragmatic hiatus. He was first seen on 18th July, 1940. Endoscopy showed that the oesophagus which was moderately dilated, contained much cloudy fluid with small quantities of milk curds. At a point 34 cm. below the alveolar margin, the oesophagus was concentrically narrowed to about 0.5 cm. The face of the stricture was bluish-white with fine markings, mostly longitudinal, converging on the constriction. The appearance was that of leucoplakia as seen in the mouth, but when the white area was touched with the instrument it oozed blood readily. A No. 26 oesophageal bougie was passed through the stricture which slowly but easily dilated up to a size 30. The bougie was not gripped tightly as by a fibrous ring but it transmitted a sensation as though the stricture were splitting. There was moderate oozing of blood and after that had been removed bright red gastric mucous membrane could be seen pouting upwards. The constricted part was then seen to be

ulcerated and covered with bleeding granulations. Its lower and lateral limits were not seen because of the bleeding, but the upper limit was easily visualised. Where before there was a neat white margin at the surface of the stricture there was now an irregular set of white processes passing down into the ulcerated area and interdigitating with bright red areas. Some of the white areas were actually isolated "like thin patches of white pain on a raw surface"(Lyall 1937). A biopsy was taken from one of the white areas and was reported on by Dr. Stanbury. It showed the superficial epithelium to be ulcerated and for the most part replaced by granulation tissue. A few nests of epithelioid cells were seen in this region. There was marked fibrosis in the floor of the ulcer involving the bundles of smooth muscle and accompanied by an infiltration of lymphocytes and plasma cells. There was no pathological evidence of malignancy.

CASE 7 - F.W., was a woman 50 years of age.

HISTORY - This patient was seen in September 1940. She complained of dysphagia in July 1936. Food seemed to be held up behind the middle of the sternum, but fluids passed down normally. When obstruction occurred she obtained relief by inducing regurgitation. She

complained of pain during the act of swallowing and when she was hungry. The former was felt high in the epigastrium and behind the lower end of the sternum and radiated through to the back between the shoulder blades. It passed off as the food entered the stomach. The other pain was similar in character, but it came on one-half to one hour after meals and was relieved by food or alkali. Three or four times she vomited as much as one half cupful of blood. She suffered in this way for six months when improvement occurred spontaneously. From early 1937 to August 1938 she was free from pain and vomiting, and while she was naturally timid and careful about her food she could eat meat. In August 1938 the symptoms returned; she vomited after every meal and often brought up bright red blood; she lived entirely on concentrated fluid foods. By September 1940 she had lost only eight pounds in weight.

EXAMINATION - Clinical examination was negative. Radiological examination as seen in Fig.63 showed no obstruction to fluid or semisolid barium in the erect position. There was a slight narrowing 6 - 7 cm. above the lower end of the oesophagus. The affected area was almost 4 cm. long, no ulcer crater was noted in it, but its wall had a crenated appearance. In the supine position the oesophagus was seen to be short and the fundus of the

Fig. 63

Case 7 - Supine. No obstruction but slight narrowing 6 - 7 cms. above diaphragm. Segment affected 4 cms. long. No crater but crenated margin seen. Gastric pouch filled.

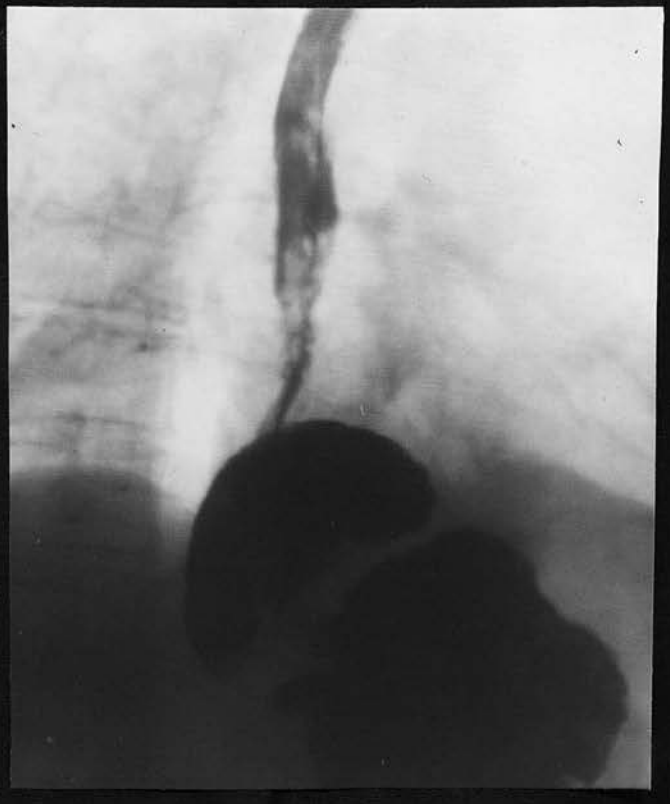


Fig. 64

Case 7 - Same patient, supine, after dilatation. Mucosa irregular but no evidence in favour of malignancy.

stomach was herniated into the mediastinum. Regurgitation of barium readily occurred when the patient lay on her right side. No ulcer crater was seen. Endoscopy was performed. At a point 26 cm. from the alveolar margin an area was found on the posterior oesophageal wall showing superficial ulceration with finger-like processes and islands of pale white mucous membrane resembling leucoplakia. The ulceration extended down to 32 cm. at which level it completely encircled the oesophagus, but at no point did it appear to be deep. Just beyond 32 cm. the gastric mucous membrane was seen. There was moderate oozing of blood as the instrument passed through the ulcerated, constricted region. As the instrument was being withdrawn a white nodular area was seen on the posterior wall which was very suggestive of neoplasm. Three biopsies were taken, one at 32 cm. from the anterior wall, one from the edge of the ulcer at 30 cm. and one from the pale nodular area seen on the posterior wall. Professor Stewart reported that the first of these showed squamous epithelium and infiltration by inflammatory cells, mainly polymorphonuclears; some of the epithelial cells were irregular in size and shape with hyperchromatic nuclei and prominent nucleoli suggestive of early malignant change. The second biopsy consisted of a piece of

squamous epithelium which at one point was thinned, sending down active-looking finger-lie projections; below this were small clusters of tumour cells infiltrating the underlying muscle. The third biopsy showed beneath the epithelium, nodules of neoplastic tissue almost certainly malignant and possibly epithelial. The interpretation of the histological picture was extremely difficult and Professor Stewart kindly submitted the sections to Professor J. Shaw Dunn who agreed that there was substantial evidence of ulceration and that the process in the muscle layer was probably diffuse carcinoma. A review of all the evidence in this case suggested that it was probably one of simple ulceration and short oesophagus with supervening malignant change. The immediate response to dilatation was satisfactory. The appearances of the oesophagus and the clinical condition of the patient in September 1941, however, raised considerable doubt about the validity of the microscopical diagnosis of malignancy. The course pursued suggested that this too was an uncomplicated case of peptic ulcer and short oesophagus. (Fig. 64).

CASE 8 - E.K. was a man, aged 63 years.

HISTORY - The patient was seen in July 1941, and for three months before this he had complained of increasing

dysphagia. Fluids would pass down without difficulty but solids were felt to be held up at the root of the neck. The obstruction seemed to be caused by mucus rising up from the stomach to meet the food. He would try to wash solids down with water, they might pass on or be regurgitated. He had not lost weight and complained of no respiratory symptoms. Previous to the dysphagia he had not suffered from indigestion.

EXAMINATION - He looked well and no abnormalities were found on clinical examination. Radiological examination (Figs. 65 and 66) showed that the oesophagus was dilated as far down as the ninth dorsal vertebra, at which level there was a persistent narrowing with an irregularity of the wall extending upwards and on the left. About 5 cm. below the top of the constriction gastric mucous membrane was seen. Besides being drawn up by the short oesophagus, the stomach formed a typical para-oesophageal hernia. Oesophagoscopy was performed 8th July, 1941. There was some dilatation of the upper part of the oesophagus. At 28 cm. the wall was covered with patches of filmy exudate and when these were wiped off with a swab an intensely red oozing surface was found beneath. A biopsy was taken from one of these areas and Dr. Horne reported that for the most part it consisted of submucosa covered at one point by a

Fig. 65

CASE 8 - Erect position showing persistent narrowing of the oesophagus opposite the ninth thoracic vertebra with dilatation above.

Fig. 66

CASE 8 - Semi-erect position showing gross irregularity in the stenosed area with shortening of the oesophagus and a typical hiatus hernia. The crater is seen in profile.

fibrinous necrotic layer; numerous bundles of muscle were seen and the fibrous stroma between them was infiltrated by lymphocytes and plasma cells; there was a small area of granulation tissue which was infiltrated by polymorphonuclears, lymphocytes, and a few eosinophils; at the edges of the muscle bundles some of the cells showed a basophilic (? degenerative) process very similar to that seen in Case 7. When the oesophagoscope was passed beyond this area the wall became more rigid and more irregular until at 32 cm. further progress was impossible. At this point the wall became grossly irregular particularly on its posterior aspect, where large mammilliform projections of leathery consistency were seen. One of these was removed for histological examination. The appearance suggested an advanced degree of leucoplakia with superadded malignant change. Histological examination, however, proved it to be simple. Dr. Horne reported that the bulk of the biopsy was covered by squamous epithelium showing marked leucoplakic changes; at one end the epithelium disappeared and was replaced by a chronic ulcer; the floor of the ulcer was of granulation tissue and beneath this were changes exactly similar to those described in the first biopsy.

The subsequent course, operation notes and post-mortem details are to be found in Appendix B.

CASE 9 - S.E.G. was a woman, aged 69 years.

HISTORY - This patient was seen on 1st October, 1941 when she was unable to take even fluids. For many years she had had a dull aching pain behind the lower end of the sternum during the time she was eating. This did not go into the back or radiate into the abdomen. She had complained of dysphagia for three years. In the beginning the obstruction was to solid food only, which seemed to lodge at the lower end of the sternum, rise up gradually into her throat, and make her vomit. There were periods of remission when food seemed to pass unimpeded. For two years she had had similar intermittent difficulty with fluids and for two weeks there had been complete obstruction. During the last year of the illness she had not felt the original pain behind the sternum which had been her first symptom. She had lost twenty-eight pounds in weight.

EXAMINATION - She was a thin woman in whom no abnormal physical signs could be found. Radiological examination (Figs. 67 and 68) showed that the oesophagus was somewhat dilated down to a point 7.5 cm. above the diaphragm, where it narrowed smoothly but abruptly into a stricture 0.5 cm. long. The channel then widened sharply into a segment

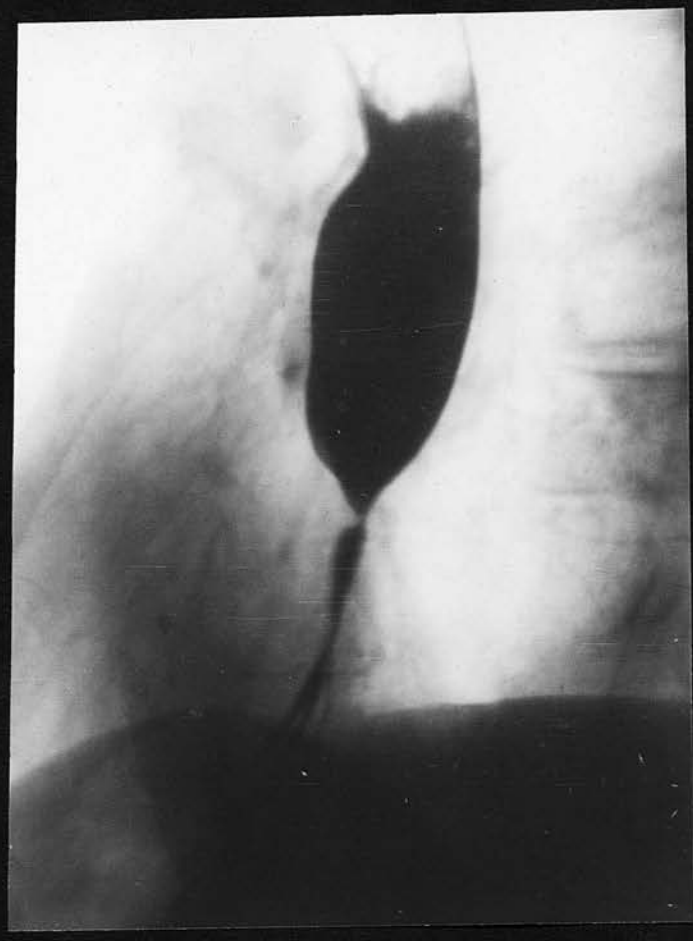


Fig. 67

CASE 9 - Erect position, showing oesophageal stenosis with dilatation above. Coarse folds can be seen in segment below.

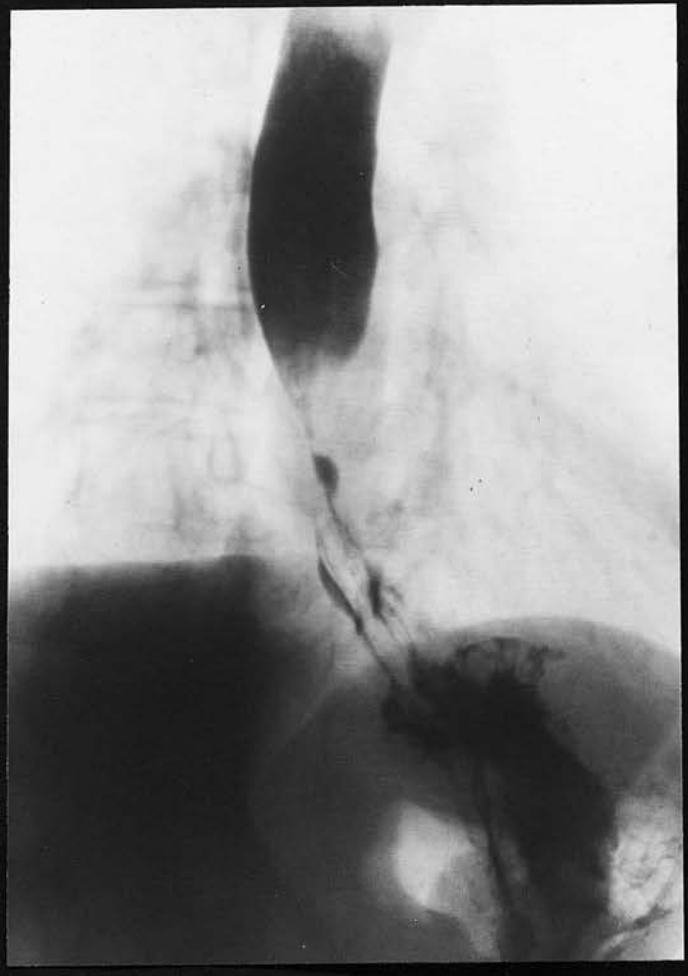


Fig. 68

CASE 9 - Supine position; gastric pouch now demonstrated but incompletely filled and ulcer crater visible.

containing broad coarse rugae. This was obviously a gastric pouch. An ulcer crater could be seen on the left lateral aspect of the oesophagus. Endoscopy was done 7th October, 1941. The oesophagus was a little dilated but the mucous membrane looked normal. At a point 32 cm. from the alveolar margin it was concentrically narrowed and was apparently completely obstructed. There was a very small depression containing a few bubbles of mucus to indicate the position of the channel. A series of bougies was passed through the stricture. It dilated with some splitting, much as the anus is dilated for a fissure and, indeed, when half dilated the picture was very like that seen in the anus with a shallow diamond-shaped ulcer in the middle line posteriorly. After passage of the largest bougie the irregular lower margin of the oesophageal mucosa could be seen and below this was a deep red bleeding zone. Two biopsies were taken from the posterior and lateral walls of the narrowed region, and it was then cauterised with some twenty per cent silver nitrate. Dr. Horne reported that one of the biopsies consisted of a portion of inflamed gastric mucous membrane. The second piece was from a chronic ulcer whose floor consisted of dense fibrous tissue with a more cellular

superficial layer infiltrated by polymorphonuclear leucocytes; at one end of this was a small piece of squamous epithelium greatly distorted by polymorphonuclear infiltration.

The subsequent progress has not been satisfactory for the patient has required several oesophagoscopies and dilatations of the stricture.

CASE 10 - This patient J.T.C. was a 50-year old man.

HISTORY - When first seen 26th October, 1941 he had been suffering from indigestion for three years. The symptoms were those of a duodenal ulcer. He had been in the hospital in August 1941 when a typical chronic duodenal ulcer had been found radiologically. While under treatment he had apparently bled freely from the ulcer and required blood transfusion. His teeth, which were very septic, were not treated. At about the same time, he used to complain of gas rising up from his stomach and causing soreness deep in the neck, but this was the only symptom referable to the oesophagus until late August 1941, when some chicken was held up and regurgitated. From that time he noticed a soreness behind the lower end of the sternum as food passed down and a gradually increasing dysphagia for solids.

EXAMINATION - There was marked pyorrhoea but no other abnormalities. The radiological examination (Figs. 69 and 70) showed that the oesophagus tapered smoothly to the face of a stricture 8 cm. above the diaphragm. The stricture seemed to extend for about 5 cm. to end in a small pouch of stomach which was pulled up into the mediastinum. The walls of the stricture were irregular but no ulcer crater was demonstrated. Oesophagoscopy showed that at 34 cm. there was a funnel-shaped narrowing. Immediately above this the mucous membrane showed much superficial inflammation with bright pink areas interspersed with bluish-grey zones. At the face of the stricture the lumen formed a lateral slit 2 cm. by 5 cm. When a bougie was passed it was gripped tightly by dense fibrous tissue and caused moderate oozing of blood. After dilatation the appearances were of a dense ringlike fibrous stricture above a deep maroon, irregular area of ulceration, which seemed to be about 2 cm. to 3 cm. long. Below this a glimpse of the gastric mucosa was obtained. Two small biopsies were taken, one from the face of the stricture and one from the inside of the stricture. The ulcer was painted with twenty per cent silver nitrate. Dr. Horne reported that the first biopsy consisted of normal squamous epithelium and some fibrous tissue infiltrated with lymphocytes. The second was very

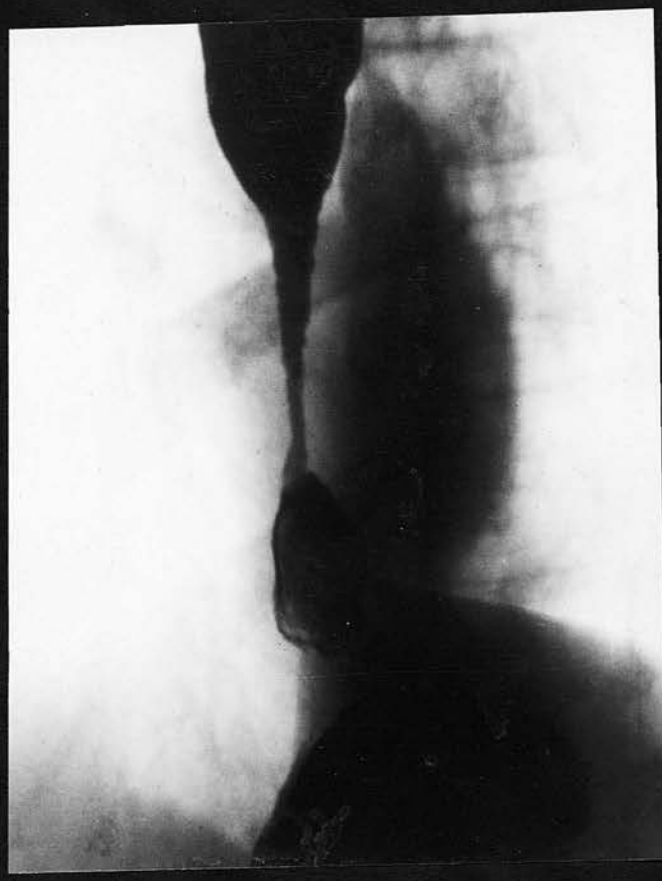


Fig. 69

CASE 10 - Supine position revealing crenated appearance of stenosed segment. Gastric pouch filled.

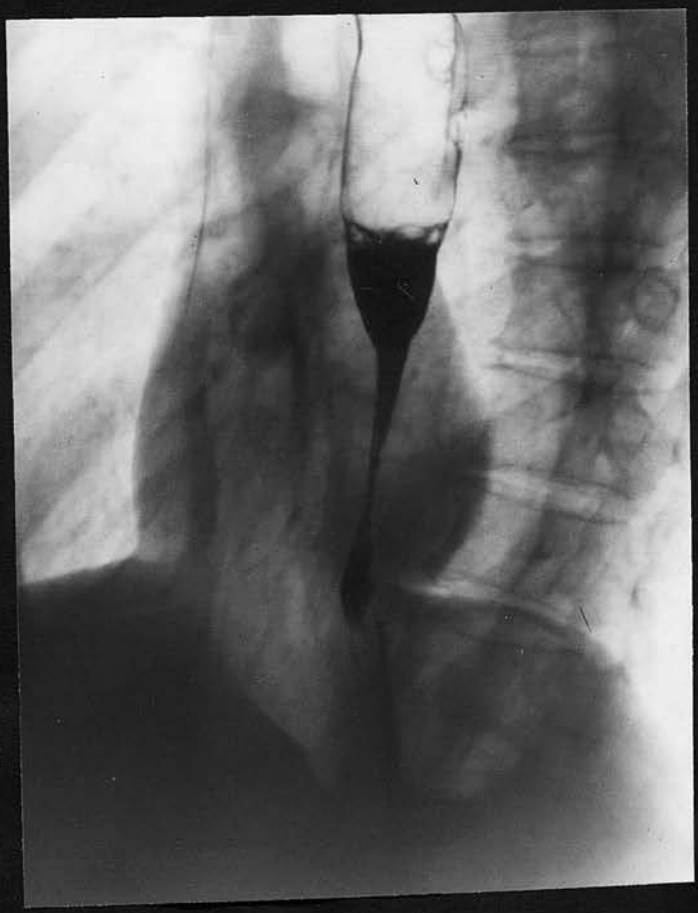


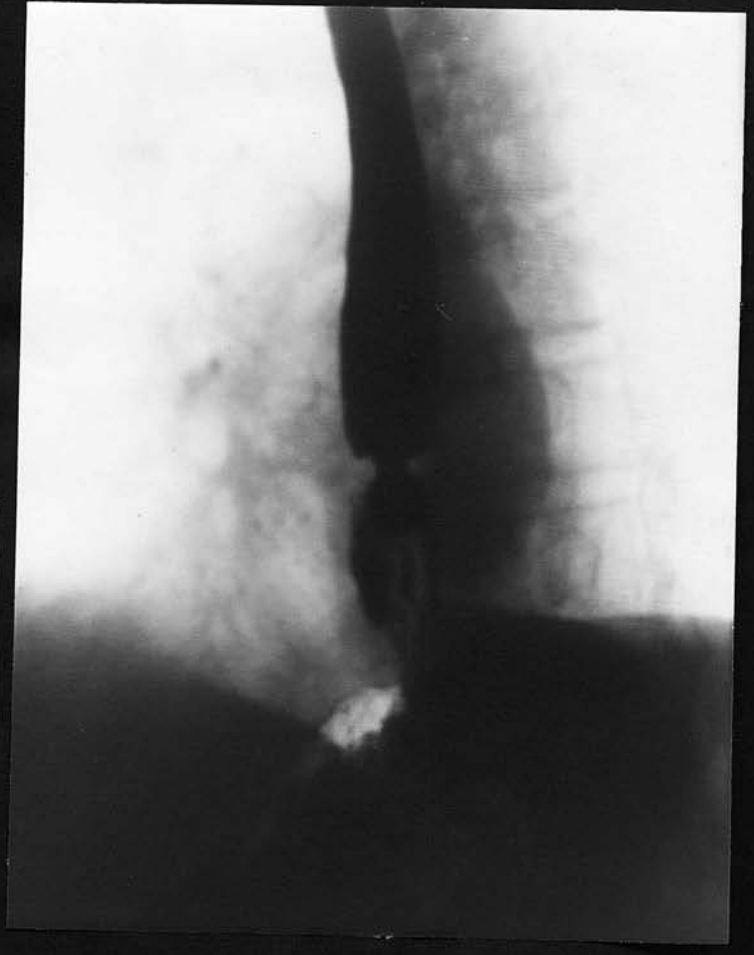
Fig. 70

CASE 10 - Erect position showing gradually tapering stenosis with dilatation above.

Fig. 71

Fig. 72

Fig. 73



Case 11 - Fig. 71. Erect position shows stenosis 3 cm. above left dome of diaphragm. Gastric pouch partially filled. Fig. 72 shows pouch mucosa ? gastric. Fig. 73 supine. Varicosity of folds in pouch evident.

vascular granulation tissue heavily infiltrated by lymphocytes, polymorphonuclears and eosinophiles, overlying a zone of dense fibrous tissue.

The subsequent progress of this patient has not been satisfactory. The recurrent dysphagia, however, is temporarily relieved by oesophagoscopy and dilatation.

CASE 11 - J.F., male, aged 67.

HISTORY - The patient was quite well until three weeks before admission when he complained of something solid sticking in his throat while taking a meal. He felt pain at the lower end of the sternum but the food seemed to stick at a higher level. Water did not move it so he made himself vomit and brought up some sticky mucus after which he finished his meal without incident. Since then he has had intermittent difficulty in swallowing solids. His appetite remained good. He suffered no loss of weight.

EXAMINATION - Radiological examination revealed a shallow ulcer in a short narrow segment of the oesophagus 3 cms. above the left dome of the diaphragm. Below this segment the mucosa was thickened and gastric in type (Figs. 71-73). A diagnosis of peptic ulceration and partial thoracic stomach was made and this was confirmed by oesophagoscopy when an acute ulcer was found at 40 cms. After dilatation a second

ulcer was seen on the posterior wall at a slightly lower level. A biopsy taken showed only gastric mucosa. The subsequent progress has been satisfactory.

CASE 12 - F.G., male, aged 65.

HISTORY - The patient had complained of dysphagia for three months which had gradually become worse. He had been reduced to taking fluids and minced solids. These had to be taken slowly and anything bulky seemed to stick in the throat. He suffered no pain and there was neither regurgitation of food nor nocturnal discomfort.

EXAMINATION - On radiological examination the oesophagus was found dilated above a short stenosis approximately 10 cms. above the hiatus. A deep ulcer crater was displayed in the anterior wall of the stenotic zone. Immediately below a typical gastric pouch was found. The rest of the stomach and duodenum appeared normal so a diagnosis of peptic ulceration of the oesophagus and a partial thoracic stomach was made (Fig. 74). This was confirmed by oesophagoscopy and after dilatation of the stenosis typical gastric mucosa was seen. A section from the ulcer was reported by Dr. H$_o$rne as an indefinite superficial ulceration with extensive submucous infiltration. No evidence of malignancy was found.

Subsequent progress was unsatisfactory and a jejunostomy was performed. Six months later the jejunostomy had closed,

Fig. 74

Case 12.

Supine film showing high stenosis with deep ulcer crater in the centre. Wide dilatation of oesophagus above. Gastric pouch filled below.

the patient was eating well and the oesophagus appeared to have healed. The large gastric pouch in this case was almost certainly of congenital origin.

CASE 13 - E.A.C., female, aged 75.

HISTORY - Three years ago she first noticed difficulty in swallowing food. This varied from time to time but for the last four months only fluids had been swallowed. Swallowed food seemed to stick at the lower end of the sternum bringing on a feeling of compression. Sometimes she felt a severe pain across the front of the chest passing round to the left shoulder. The pain frequently woke her at night. It was not in any way affected by posture. There had been slight loss of weight recently.

EXAMINATION - On passing the oesophagoscope an obstruction was reached at 38 cms. and fluid retention was very marked. A bougie was passed through the stricture and this was followed by much oozing which obscured the field. A piece of tissue from the stricture was removed for biopsy. Dr. Horne reported that there was no evidence of a tumour but he found some loose fibrinous material in one part of the section which appeared to be debris from the floor of an ulcer. Three days later the patient was examined radiologically and on swallowing semi-solid barium a slight, persistent narrowing was observed about 6 cms. above the diaphragm. At this point a small niche was seen suggestive of an ulcer.

The mucosal folds below showed the characteristic gastric pattern and a partial thoracic stomach was demonstrated. (Fig. 75).

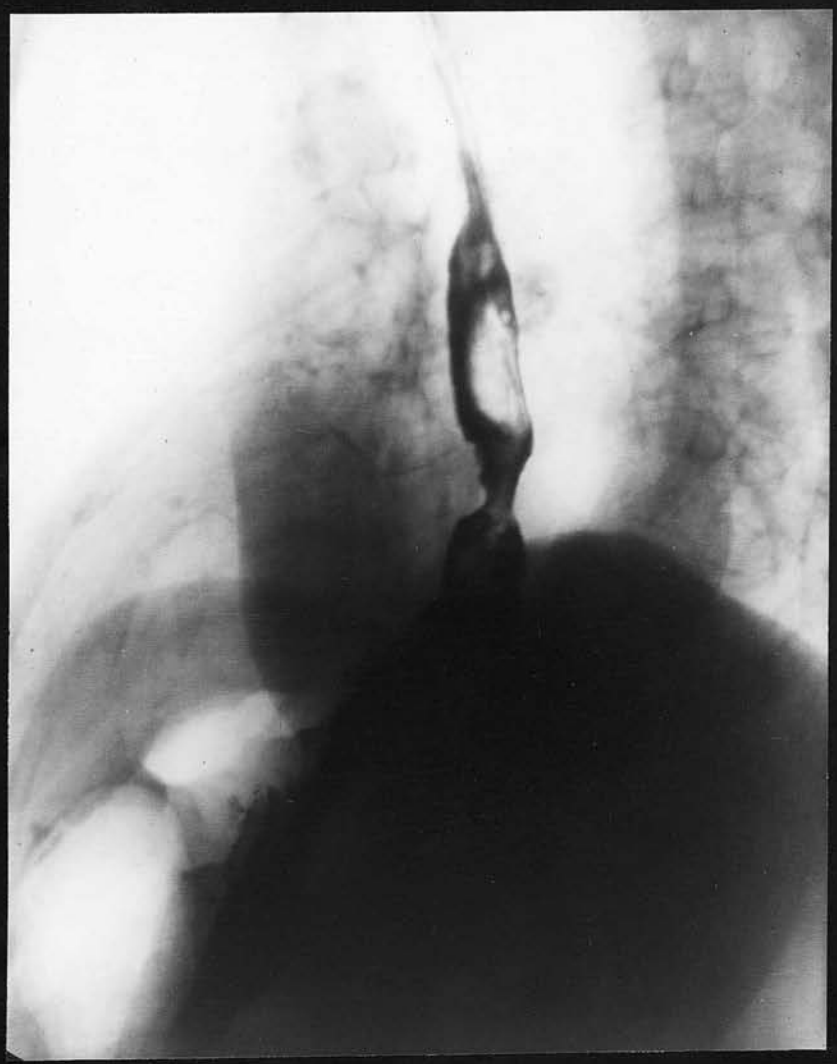
For a short time after dilatation the patient showed considerable improvement but she gradually relapsed and two years later gastrostomy was performed. Swallowing has since improved considerably but the gastrostomy has not yet been closed.

CASE 14 - A.S., male, aged 68 years.

HISTORY - For the past sixteen years he had complained of occasional indigestion after meals. About a year before admission the indigestion became more frequent and eight weeks prior to admission he began to have a severe epigastric pain coming on a quarter to half-an-hour after meals. He went to bed and was put on an ulcer diet but for the past ten days he had been unable to drink milk or any other fluid - these being returned as a projectile vomit.

EXAMINATION - Radiological examination was first carried out and the oesophagus presented a long, smooth funnel-shaped narrowing which commenced about 5 cms. below the bifurcation of the trachea. No ulcer was visible and the thickening of the wall suggested infiltration due possibly to malignancy. On account of its situation an alternative diagnosis of peptic ulceration was suggested. Insufficient barium passed

Fig. 75



Case 13.

Supine film. Examination made after dilatation. Stenotic area seen with small ulcer crater anteriorly. Below stenosis is short gastric pouch.

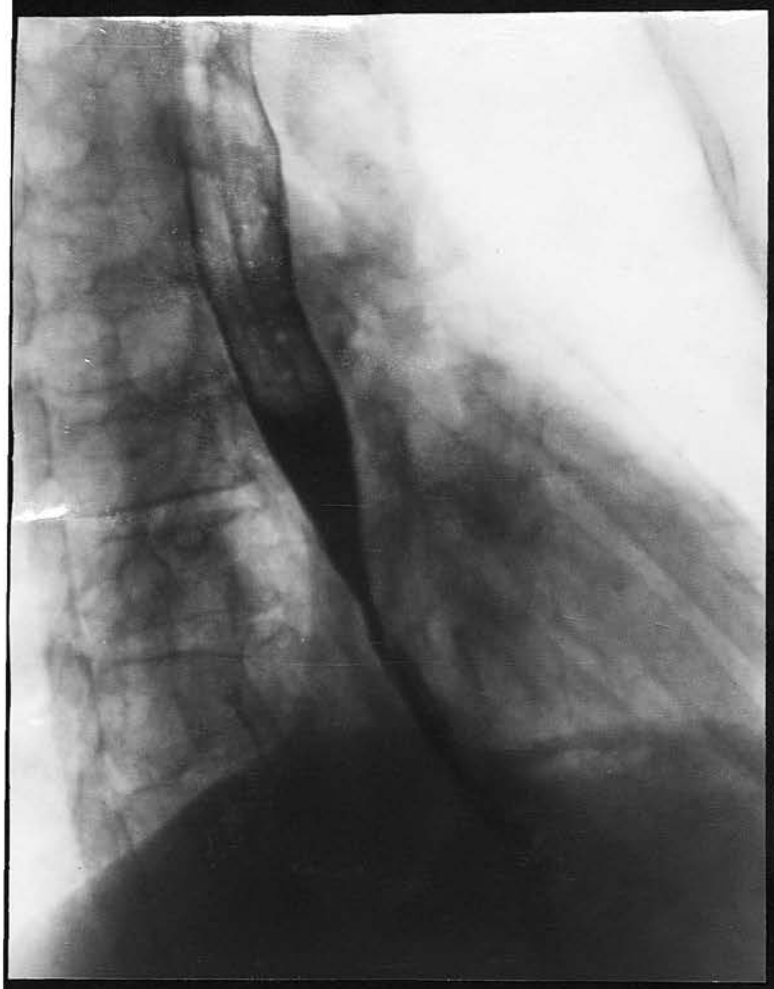


Fig. 76

Case 14 - Erect, after dilatation. Oesophagus tapers down to stenosis 2½" above diaphragm.

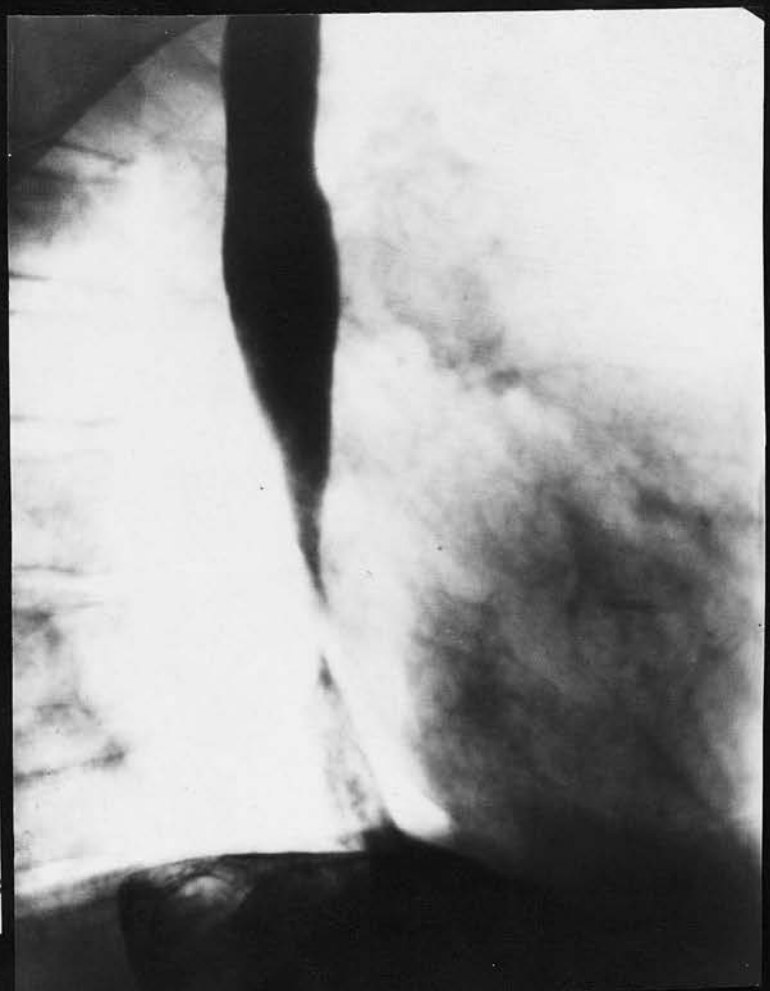


Fig. 77

Case 14 - Same patient, supine. Oesophagus outlined. Coarse folds seen in lower segment.

through the stenosis to flood the segment below it so the combination of lesions was not established. Oesophagoscopy was performed the following day and the oesophagus was found narrowed down to the diameter of a crow quill. There was an intense oesophagitis above this area. After some difficulty bougies were passed and then the stenosis was easily and rapidly dilated up to size No. 30. No evidence of malignant tissue was seen in the lumen.

No biopsy was taken because the patient collapsed under pentothal. A few days later another barium swallow was given and the tapering stricture about 2 cms. long was seen well above the diaphragm. Below the mucosa had the coarse appearance of gastric rugae. No typical flooding of the herniated pouch occurred during the examination but the diagnosis of oesophageal peptic ulceration with a partial thoracic stomach was made (Figs. 76-77). The subsequent course of this patient was unsatisfactory. Swallowing was largely restricted to semi-solids and a gastrostomy was advised. This was refused by the patient and when he was seen a year later he claimed to have put on a little weight and stated that he was swallowing better though restricted to semi-solids.

CASE 15 - H.S., male, aged 55 years.

HISTORY - Six years previously the patient had begun to suffer from epigastric pain. It was in no way related to meals but he noticed that fatty foods made it worse. Some months later he began to vomit and was admitted into hospital for investigation. He was thought to be suffering from peptic ulceration of the stomach or duodenum but no abnormality was found. This put his mind at ease and he felt much better. He remained well for five years and then the vomiting returned. Food would come straight back five to fifteen minutes after a meal. At no time, however, did he complain of pain. He stated that he coughed up a lot of phlegm which seemed to come up from the epigastrium and behind the sternum. He lost a little weight. This continued for about nine months when suddenly three weeks ago he was unable to swallow anything. He was examined by another radiologist (Dr. Wall) who found an even narrowing of the oesophagus about mid-way between the bifurcation of the trachea and the oesophageal hiatus. The lumen was only one or two millimetres in diameter and the obstruction appeared marked. The oesophagus above the stricture was only a little dilated.

Following the examination, however, he swallowed fluids fairly well. Two weeks later he was oesophagoscoped and at 36 cms. the oesophagus was found to be intensely inflamed. Between 38 and 39 cms. the lumen seemed to be completely obstructed and the mucosa was an intense red. Bougies were passed and the stricture was dilated. There was no evidence of malignancy and the appearances were those of peptic ulceration. A second oesophagoscopy was performed ten days later. The same inflammatory changes were observed. Blood oozed freely and erosion of the mucosa was seen. At 40 cms. the stricture had contracted again but was readily dilated. On this examination Mr. Allison considered the condition to be an intense oesophagitis above a healed ulcer with stenosis.

The patient was given a second barium swallow two days later and this time there was but little delay. No ulcer was seen and below the slight narrowing the mucosal folds became coarse and appeared to be of gastric origin but a gastric pouch could not be demonstrated by posture (Figs. 78-79). This examination was followed by another oesophagoscopy five days later and a biopsy was obtained from the ulcerated area. This was reported as being most likely inflammatory tissue rather than neoplastic.

The immediate response was so good that within two months the patient gained a stone in weight. In this case the improvement has been maintained.

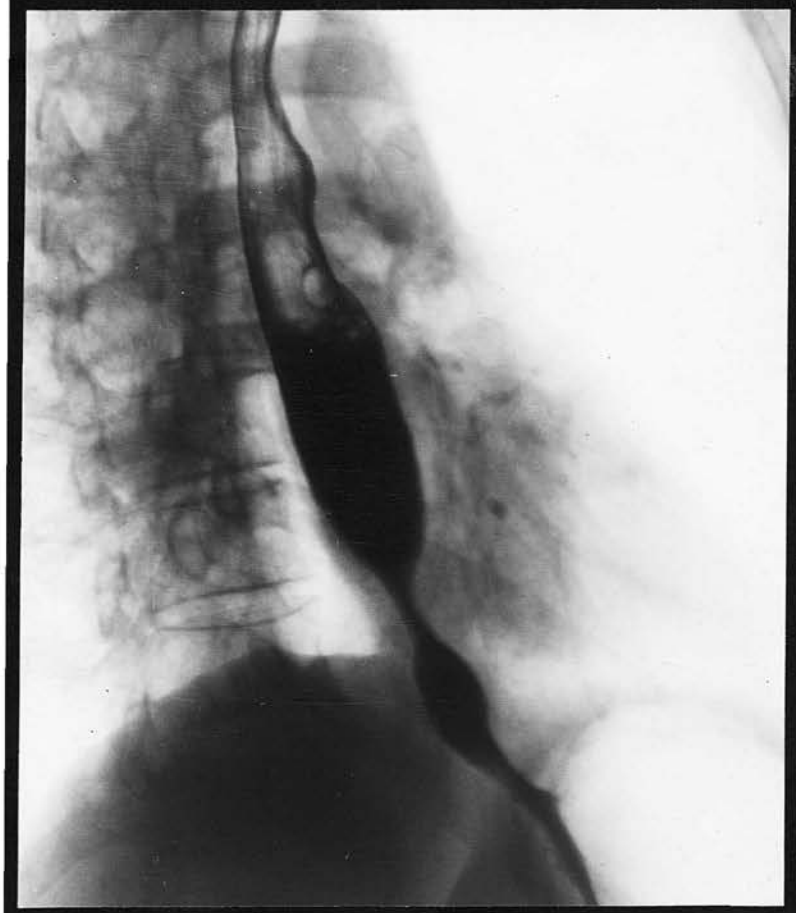


Fig. 78

Case 15.

Erect position, right oblique, after dilatation. Stricture demonstrated, but no ulcer.

Fig. 79

Same case, left oblique. Gastric folds are seen running above the diaphragm.

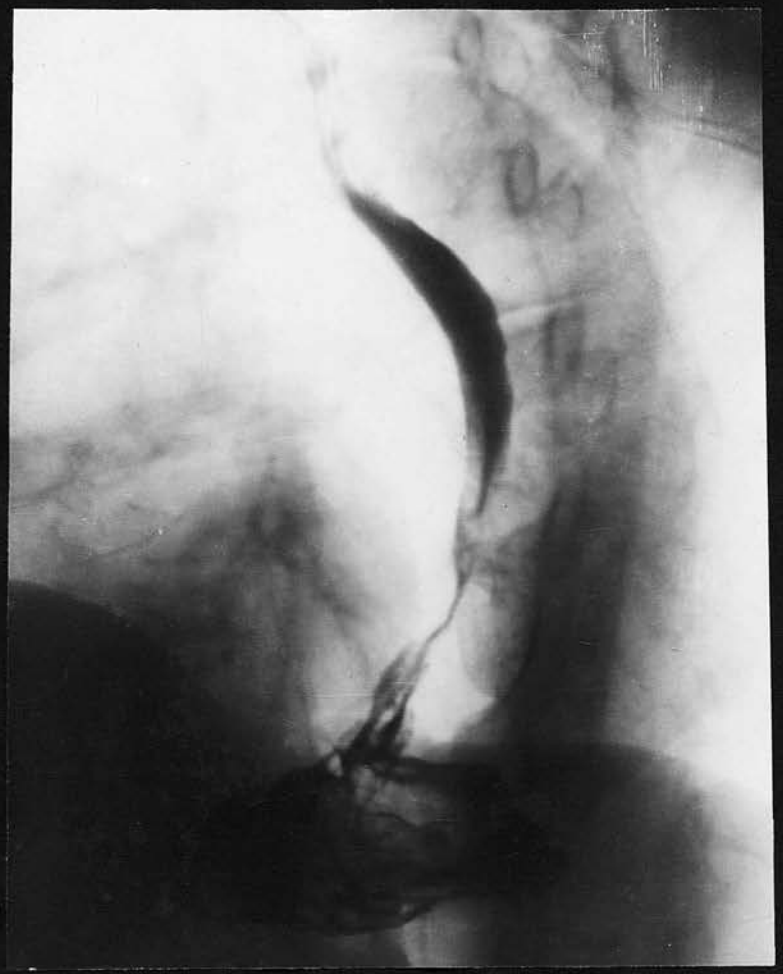


Fig. 80

Case 16 - Semi-supine, after dilatation. Short stenotic area seen with gastric pouch below.

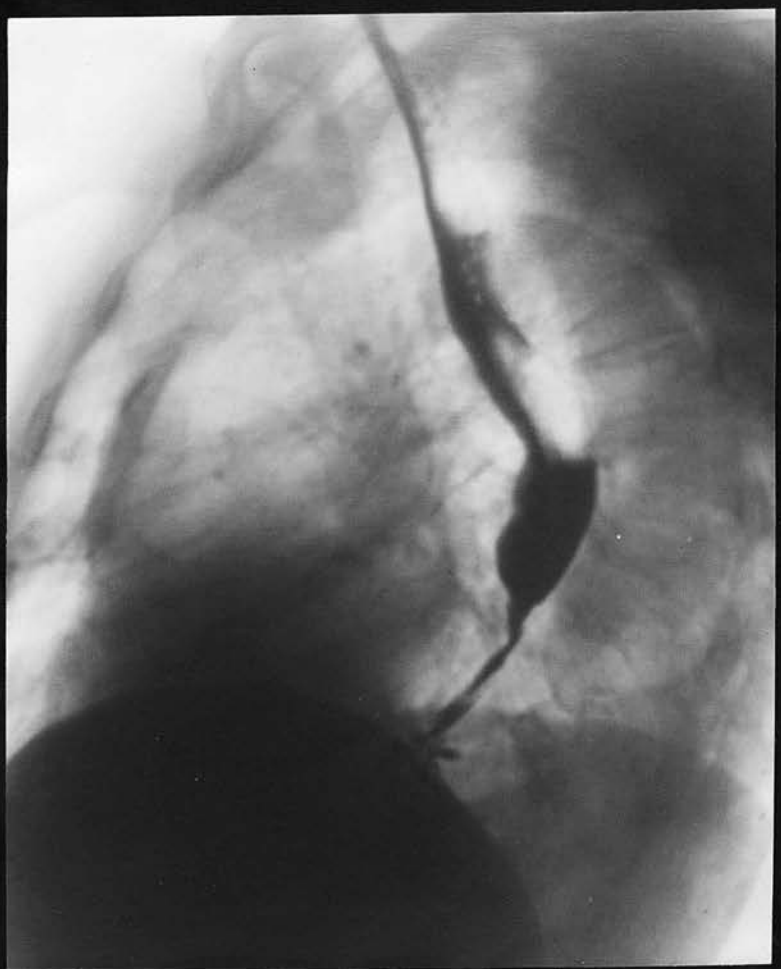


Fig. 81

Case 16 - Erect, after dilatation. Stenotic area 1" long. No crater seen. Mucosa below has gastric appearance.

CASE 16 - E.R., female, aged 67 years.

HISTORY - The patient stated that she had been unable to eat or drink during the past three weeks. On enquiry she said that for fifty years she had suffered from 'stomach trouble.' When she swallowed food it seemed to stick behind the sternum and she discovered that taking some bicarbonate of soda would relieve her. The food eventually passed on and vomiting never occurred. She thought fat meats slipped down best. Three weeks ago there was a sudden and apparently complete stoppage. She had never had any pain.

EXAMINATION - On radiological examination a complete obstruction in the oesophagus was found about 5 cms. above the diaphragm. Some irregularity of the walls was noted and the appearances suggested malignancy. The following day barium was still present when the oesophagoscope was passed. At 30 cms. the walls became very red and the lumen was completely obstructed. The stenosis dilated readily and some oozing occurred. The appearances were those of a simple ulceration. On repeating the barium swallow after dilatation, fluid barium passed freely down the oesophagus. A stenotic segment about 2.5 cms. long was revealed at the site of the previous obstruction but it dilated moderately well allowing the bolus to pass. Below this segment the mucosa was of the gastric type but no typical filling of a

(Figs. 80 and 81)

herniated pouch was obtained. Three weeks later oesophagoscopy showed that some narrowing had recurred and dilatation was carried out. Some leucoplakia and superficial ulceration were observed and a biopsy was taken. This was reported by Professor Stewart to be currettings from a simple chronic peptic ulcer of the oesophagus.

CASE 17 - T.A.H., male, aged 69 years.

HISTORY - For six years the patient had been troubled with indigestion. After the first few mouthfuls of food he was apt to vomit, bringing up thin white frothy material which did not always contain food. Flatulence troubled him considerably. These attacks of vomiting would come on every two to three days.

Four years ago he underwent an operation for a peptic ulcer. The site and type of operation were not known. Subsequently he remained well for a year but during the last three years his symptoms had returned and had become progressively worse.

Seven days before admission to hospital he vomited about two pints of blood. On admission he complained of an aching pain across the left lower chest and umbilical area, but the pain had no relation to meals. His appetite had always been good and he had lost little if any weight.

EXAMINATION - On radiological examination a narrowing was noted at the lower end of the oesophagus together with a persistent niche suggestive of a peptic ulcer. Distal to the niche the mucosal folds were thick resembling gastric mucosa but a typical filling of a hiatal pouch was not obtained. The stomach and duodenum appeared to be normal (Fig.82).

A well-defined, deep, penetrating ulcer was found on oesophagoscopy at 35 cms. The crater was on the anterior wall and was filled with purulent material. The edge seemed hard but was not everted. The gastro-oesophageal margin was noted at 40 cms. A test meal was carried out in this case and showed the acid secretion to be high but within normal limits.

CASE 18 - L.A., male, aged 22 years.

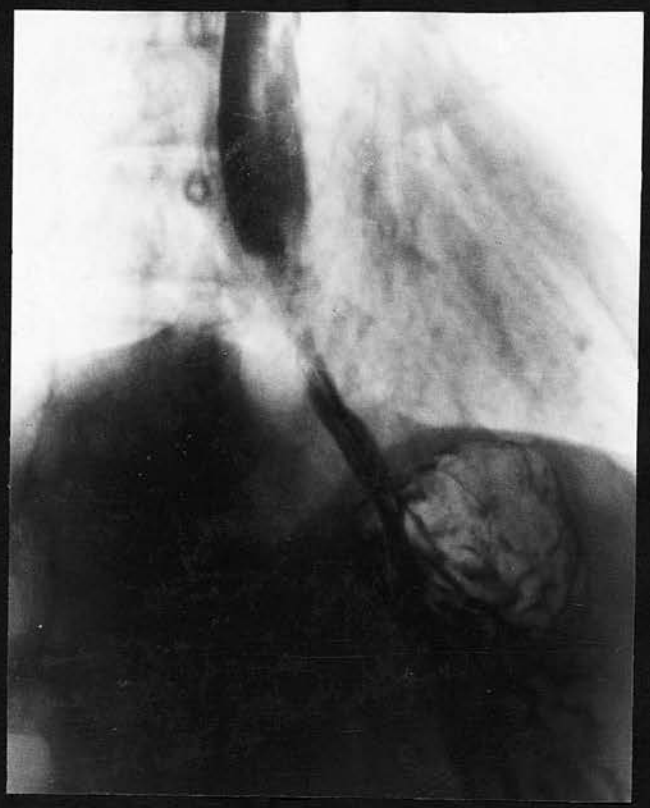
HISTORY - The patient complained of intermittent obstruction when swallowing solid foods. It was first noticed some eighteen months previously when food suddenly stuck at the beginning of a meal. After a short time this passed off and he was able to finish his meal confortably. Since then the trouble had recurred at intervals varying from a few days to a month.

He stated that the obstruction was as liable to appear at the first swallow as the last and came on without warning.

Fig. 82

Case 17.

Erect position. Pouch collapsed. Some irregularity at stenosis with persistent niche.



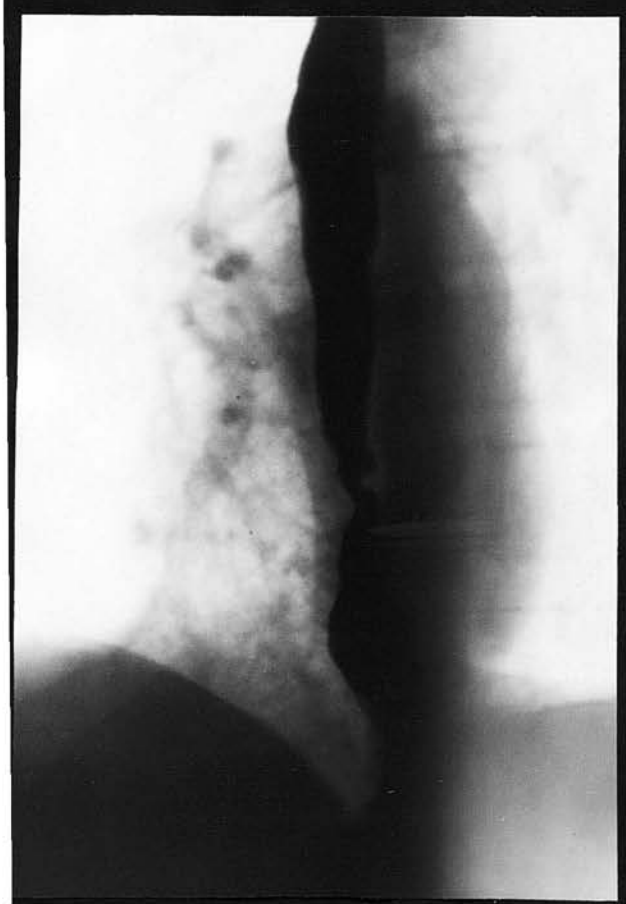


Fig. 83

Case 18 - Top left. Erect. Stenosis 1" long seen 3" above diaphragm. Below stenosis coarse rugae suggest presence of a gastric pouch.

- Top right. Supine. "Apparent" ulcer crater demonstrated.

- Lower left. Oesophagus filled showing stenosis and 'crater.'

He suffered neither pain nor indigestion. The food might come back, or occasionally he could force it down. Taking fluid drinks did not help it down. Meat was the worst article of diet for provoking the obstruction. No previous history relative to the condition was recorded.

EXAMINATION - A barium swallow showed a persistent stenosis 2.5 cms. long, situated about 7 cms. above the hiatus. A smooth-walled crater was seen on the postero-lateral wall. Above it the oesophagus was a little dilated but there was no prolonged delay in emptying. Below the narrowed segment the mucosa showed coarse rugae resembling the gastric type but no flooding of a herniated pouch was obtained. The stomach and duodenum appeared normal and a diagnosis of peptic ulceration of the oesophagus was made (Fig. 83).

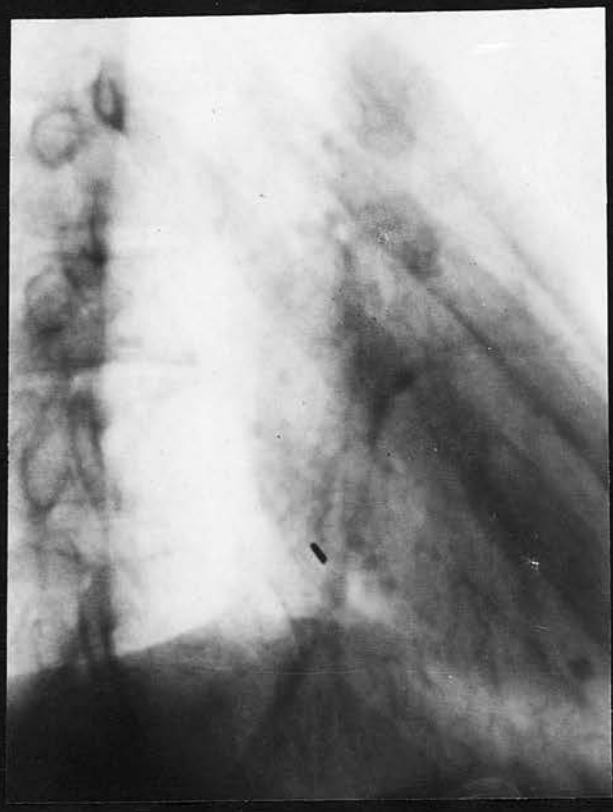
Two weeks later on oesophagoscopy no dilatation of the lumen was noted. At 35 cms. a stenosis was found with rigid, pale walls but no ulceration. A biopsy was taken from the narrowed area and a silver clip applied immediately above the stenosis. The section report was summarised as 'gastric heterotopia - no evidence of malignancy or inflammatory change.'

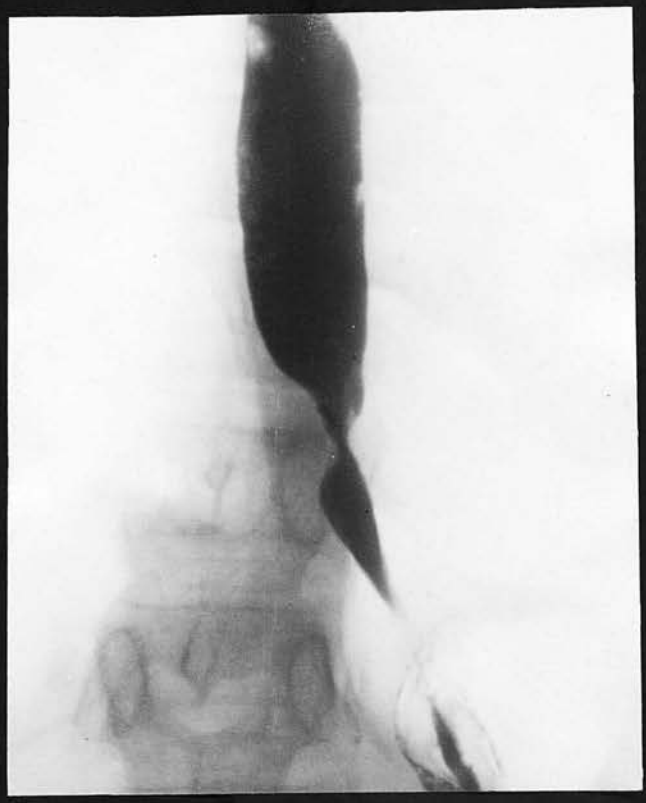
The patient was radiographed after the application of the clip and it was seen to lie 5 cms. above the diaphragm. Barium showed its position to coincide with the stenosis.

The niche regarded as an ulcer was present but not so well-defined (Fig.84).

Six weeks later oesophagoscopy was repeated and the narrowing at 35 cms. was again demonstrated. No inflammatory changes were evident and when the bougie was passed into the stricture it was felt to be in a tight fibrous constriction. Two silver clips were applied at different levels below the stricture and punch biopsies were taken. These revealed some squamous epithelium but were otherwise inadequate for diagnostic purposes. Three months later a barium swallow was carried out and the oesophagus appeared normal except for a slight stenosis. The walls were smooth and there was no evidence of a crater. A definite gastric pouch could not be filled.

This case was not straightforward and it is included to illustrate the diagnostic difficulties. The niche seen on the radiograph and diagnosed as an ulcer was not confirmed by oesophagoscopy. The original biopsy showed gastric mucosa but the second, taken from a lower level, was reported as squamous epithelium. No true gastric pouch was demonstrated although in one film the rugae strongly resembled the gastric type. Further the patient was much younger than any others in the series. It would therefore appear that this





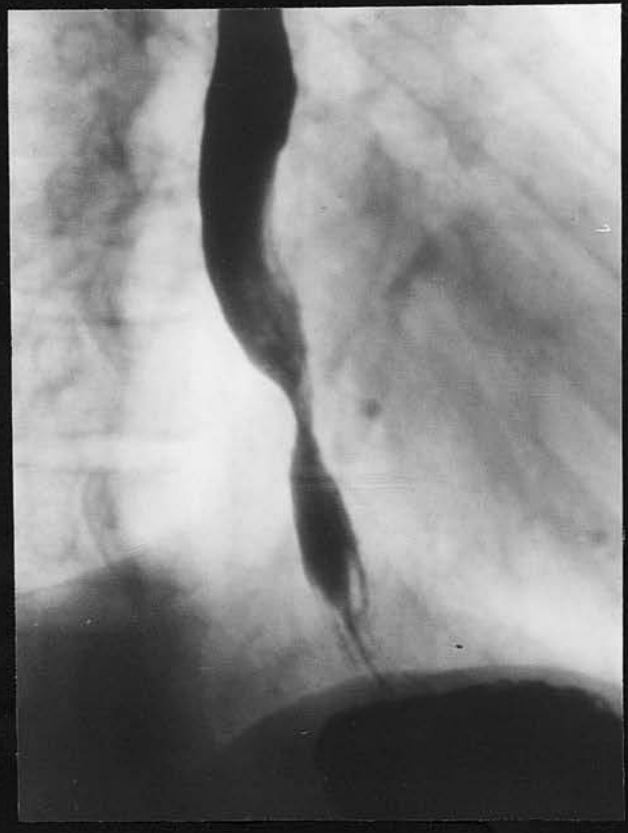


Fig. 84

Case 18 - Top left. Silver clip on mucosa at site of biopsy which revealed gastric mucosa.

- Top right. Same, after barium swallow. Clip can be seen at stenosis.

- Lower left. Four months later. No 'apparent' crater but stenosis still evident.

man may have a stricture following ulceration of an area of gastric heterotopia, but it must be remembered that the finding of squamous epithelium at a slightly lower level may be accounted for by the irregularity of the junctional mucosa which occurs at the cardia and does not exclude shortening of the oesophagus.

-----oOo-----

APPENDIX B

CASE 8 - The subsequent course of this patient was unsatisfactory and swallowing became very difficult and he suffered constant and severe pain. A jejunostomy was performed and after slight complications due to wound sepsis the patient made satisfactory progress. Five weeks later Mr. Allison explored the oesophagus and used a trans-pleural approach. The operation notes were as follows:-

"14.5.42: Open ether. Stomach tube passed and oesophagus aspirated. Intubation. Pharynx well packed off. Gas and oxygen and ether. The 8th and 9th ribs were resected on the left side and the pleura opened. The lung was free except for one diaphragmatic adhesion, which was divided. The lateral pulmonary ligament was then divided and this was greatly thickened by chronic inflammation. The mediastinal tissues contained much fat and an excess of dense fibrous tissue. The fundus of the stomach was first exposed and the mediastinal tissue then incised upwards on to the oesophagus where very dense scar tissue was encountered puckered down on to the surface of the lower end of the oesophagus. This could be felt almost like a wooden bobbin between the fingers. A catheter was passed round the stomach and another one passed round the oesophagus above the densely scarred area. This latter was not easy as there was no sharp line of distinction between the very dense scar tissue and the chronically-inflamed tissue above. By sharp dissection the cardia was freed and one or two vessels ligatured. The left phrenic nerve was then crushed and good elevation of the diaphragm seen immediately. The finger was passed through the oesophageal hiatus and an incision made through the diaphragm for about 2". Two spurting vessels were ligatured. The edges of the incision in the diaphragm were then stitched to the dense scar tissue round the lower end of the oesophagus so that the fundus of the stomach was completely below the diaphragm. There was a tendency for fatty tissue to herniate upwards on the posterior and left lateral aspect of the cardia. At this point the edge of the diaphragm had to be stitched to the dense fibrous tissue

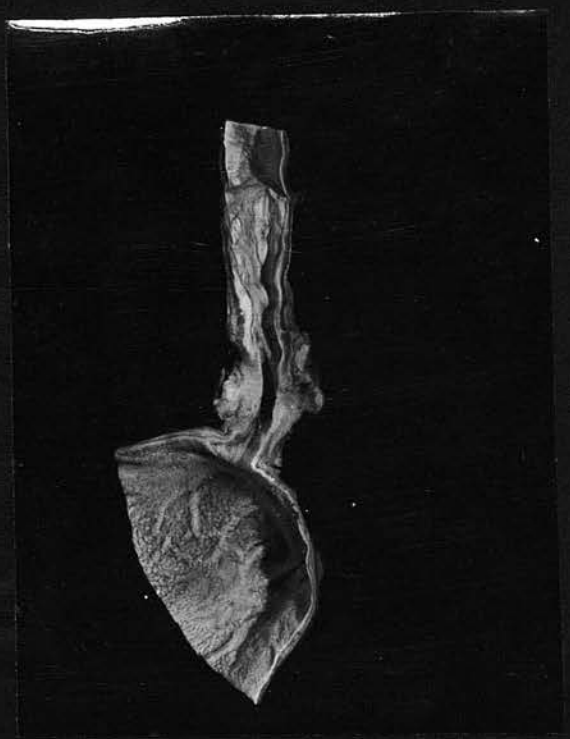
"over-lying the aorta. Both vagus nerves were divided on the lower end of the oesophagus. The drainage tube was then passed through the pleura into the para-oesophageal tissues, brought out through the anterior end of the incision and connected to an uncer-water drain. The chest was closed. Throughout the operation plasma was given."

The patient died four days after the operation and post-mortem was carried out by Professor Stewart. A photograph of the specimen is seen in Fig. 85 and the full pathological report is appended.

"In the lower third, commencing about 15 cm. from the cardia there is notable thickening of the wall of the oesophagus for a distance of about 6 cm., associated with much narrowing and distortion of the lumen. In the lower part of this lesion the appearances suggest malignant infiltration of the wall of the gullet, invading and largely destroying the muscular coat through a distance of 2 cm. The irregular mass on the outer aspect here also feels like tumour, although the appearances are not distinctive. Above this ? neoplastic zone there is great muscular hypertrophy for some 3.5 cm. together with dense fibrosis and thickening of the submucosa. In the rest of the oesophagus above this the hypertrophy is rather less, but still quite notable. In the lower part of the lesion there are local bulgings of the lumen of both the anterior and posterior walls about $2\frac{1}{2}$ cm. from the cardia. The mucosa in this situation is smooth, rather irregular and opaque. In the upper part of the oesophagus there are well-marked patches of leukoplakia.

"HISTOLOGY - A complete segment 7 cm. in length was taken through the lower end of the specimen, including the whole length of the lesion. There is ho histological evidence whatever of neoplasm. The lesion appears to be a chronic inflammatory one with ulceration and much muscular hypertrophy.

Fig. 85



Photograph of specimen (P.M. - reversed) For detailed description see report. Note there is no evidence of a herniated pouch.

"The ulceration is everywhere superficial and while it has extended down in places to the muscular coat this is nowhere penetrated to any extent, still less breached. The most interesting feature is that a patch of gastric mucosa is present no less than 5 cm. from the lower end of the specimen, and as this point coincides with a junction with squamous epithelium either this is extensive gastric heterotopia or a portion of the stomach which is pulled upwards, or possibly a combination of the two. At the lower end of the specimen a considerable portion of this gastric type mucosa is free from ulceration for a distance of about 1.5 cm. There follows a portion of ulceration which has destroyed the whole thickness of the mucous membrane and extends upwards for a matter of 2.7 cm., then another portion of surviving gastric mucosa measuring 0.8 cm.

"The lower limit of hypertrophy of the muscular wall corresponds with the lower margin of the area of ulceration and possibly corresponds with the line of junction of stomach and oesophagus. The zone of intense muscular hypertrophy measures roughly 4 cm., but in an upwards direction there is a gradual lessening of hypertrophy, whereas at the lower end there is a much more abrupt change.

"That portion of the ulcerated area which lies above the line of junction of gastric and oesophageal mucosae is characterised by complete loss of epithelium and replacement of the superficial zone of mucous membrane by a narrow layer of granulation tissue intensely infiltrated by lymphocytes, polymorphs and plasma cells. The muscularis mucosae still persists in the deeper part of the mucosa but the muscle bundles are much broken up and widely separated by infiltration with plasma cells, lymphoctes and a few polymorphs. In this situation and in the submucosa a number of capillary channels are seen to be stuffed with polymorphs while other show margination. The submucosa itself here is greatly widened and very much fibrosed, and there are many foci of lymphocutes and plasma cells, mainly perivascular in position, and indeed occasional lymphoid nodules. There is a sharp line of separation between the submucosa and the muscular coat, the line of junction being rather highly vascularised, but occasional strands of fibrous tissue pass into the muscular coat and there are patches of fibrosis within the latter. Ganglionic nerve cells of the plexus between the muscular layers are well preserved, but this zone is somewhat fibrosed, with many congested vessels, and shows slight infiltration by chronic inflammatory cells. Lymphoid foci are present here and there in the midst of the muscle. External to the muscularis there is a broad band of dense fibrous tissue with many capillary channels, some of them dilated and there is considerable infiltration by lymphocytes with, in places, many polymorphs.

"By contrast with these changes in the higher ulcerated area of what is clearly the oesophagus, the lower area of ulceration reaches down to the muscular coat. The appearance, in fact, is that of a thin layer of very cellular granulation tissue implanted directed on the muscularis, which by contrast with the higher lesion shows much breaking up of the muscle bundles by vascular connective tissue with varying amounts of infiltration by plasma cells, lymphocytes and polymorphs. The disorganisation of the muscular coat by these inflammatory changes is much more striking than higher up, but there are still well-preserved ganglionic nerve cells lying between the two main muscular coats. External to the muscularis there is a broad zone of dense fibrous tissue with engorged vessels, patchy haemorrhages, peri-neural fibrosis and on the surface a narrow zone of recent fibrinous inflammation.

"The third block taken transversely across the specimen well above the main lesion shows a thick patch of leukoplakia, together with further superficial ulceration betweeh tne leukoplakic patches. This looks active and fairly recent. Epithelium is lacking and the surface is covered by a narrow zone of intensely inflammatory tissue with very abundant polymorph leucocytes in the more superficial part. The lesion implicates the muscularis mucosae, which is hypertrophied and infiltrated by focal collections of plasma cells and lymphocytes. The underlying submucosa is oedematous rather than fibrosed. The muscular coat is practically unaffected.

"SUMMARY - No evidence of neoplasm. Simple chronic (?peptic) ulceration involving

a. a large stretch of gastric mucosa in the lower two-thirds of the specimen, and

b. a similar lesion involving the upper (oesophageal) portion. This upper area of ulceration is more superficial than the lower and it does not invade the muscular coat, which the lower does.

"DIAGNOSIS - Extensive gastric heterotopia (or in part upward displacement of gastric mucosa) with chronic but superficial peptic ulceration of both gastric and oesophageal portions."

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SUMMARY

Peptic ulceration of the oesophagus in combination with a partial thoracic stomach is described as a clinical entity.

It occurs more frequently than is generally appreciated and the differentiation from cancer is of the greatest importance.

In peptic ulceration the sex distribution appears to be about equal and seventy per cent of the cases are between fifty and seventy years old.

The chief factors in the pathogenesis are:-

(1) regurgitation of acid gastric juice into the oesophagus.

(2) Hyper-acidity.

(3) The ulcer diathesis.

Sepsis and heterotopic gastric mucosa are of less importance.

Regurgitation occurs freely when the pinchcock actions of the diaphragm and the circular muscle fibres at the cardia are lost. It is held that this happens in cases of short oesophagus and partial thoracic stomach.

The treatment is medical in early cases with a strict dietetic régime and raising the head of the bed. When obstruction is marked, dilatation may bring relief. This is often only temporary and then jejunostomy, gastrostomy, local resection or repair of the hernia myst be considered.

After care follows the same lines as that recommended for peptic ulceration in the stomach and duodenum.

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